VOLUME 79 • DECEMBER 1959 • NUMBER 6

A.M.A. ARCHIVES OF

SURGERY

INDEX NUMBER

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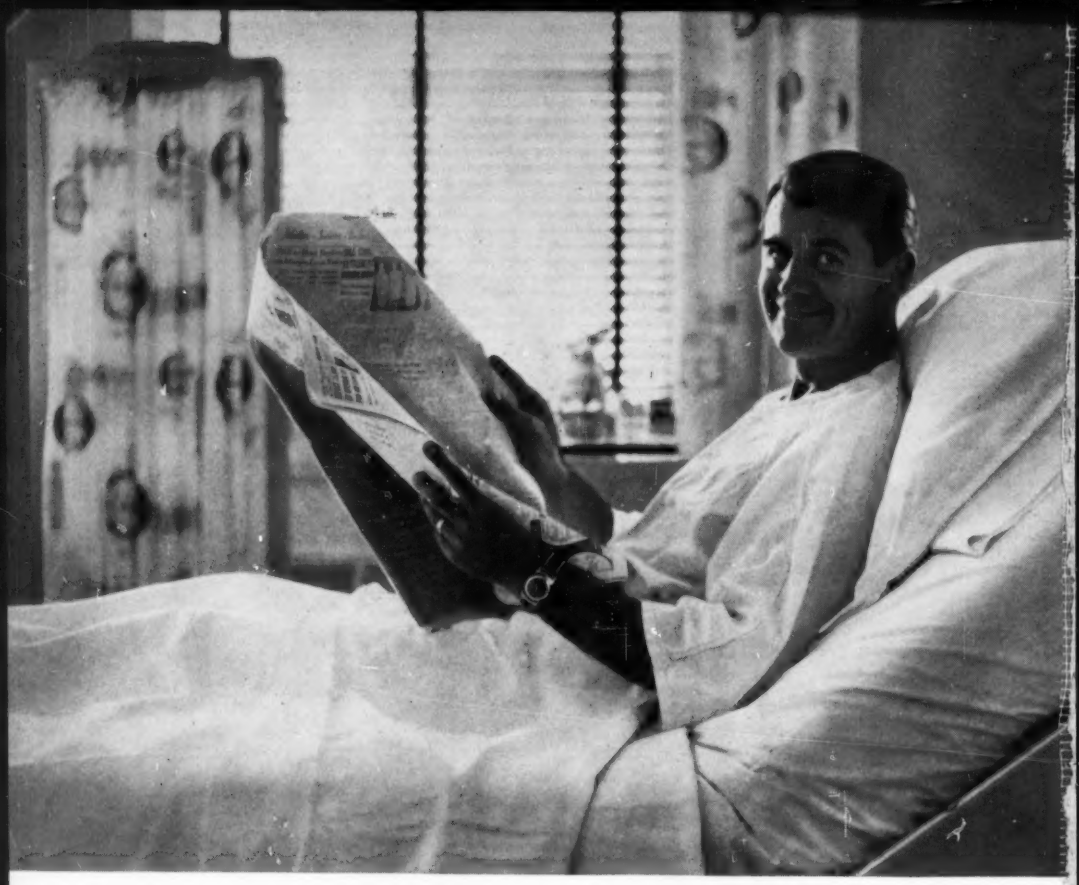

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ARCHIVES OF SURGERY

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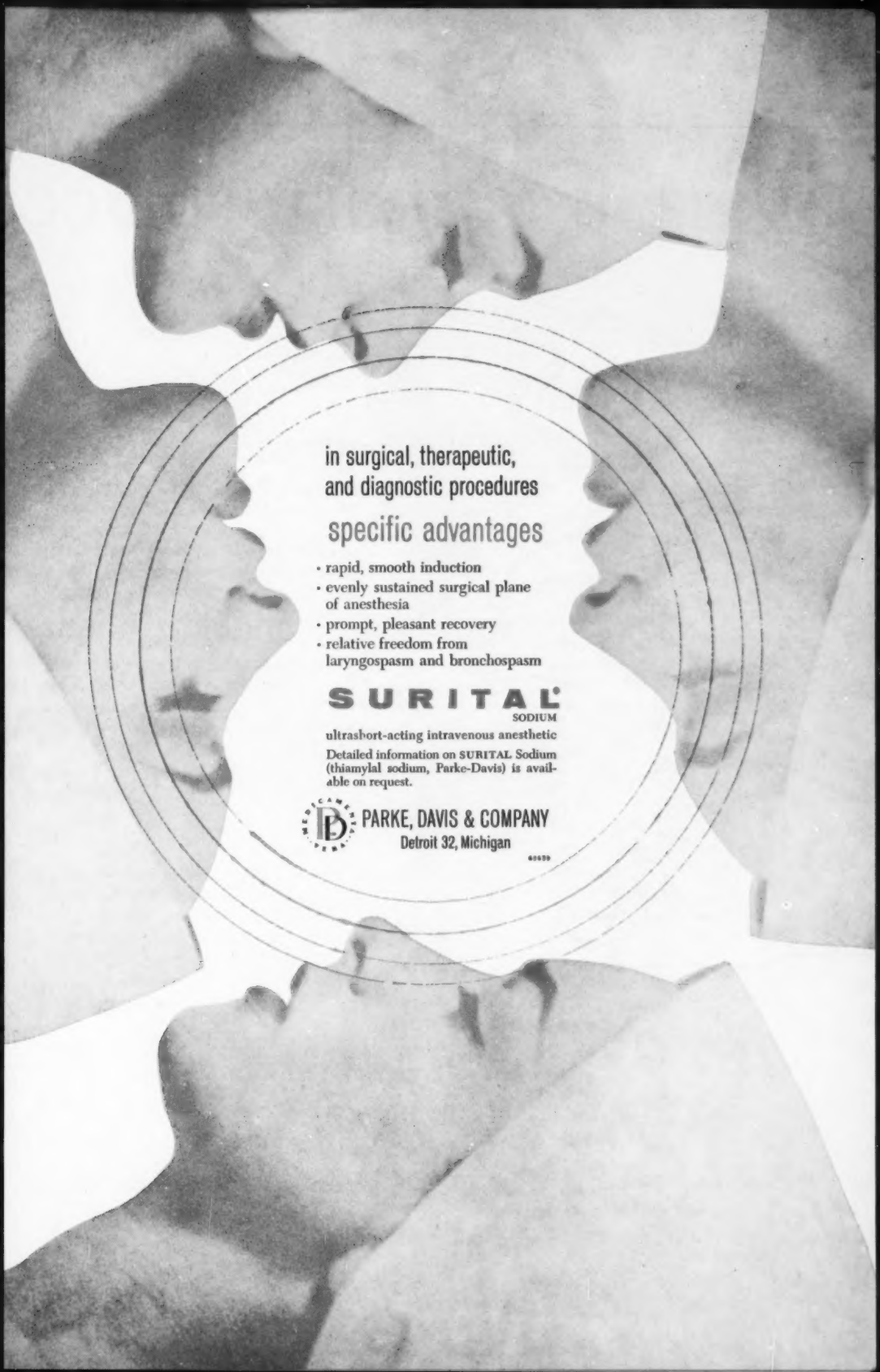
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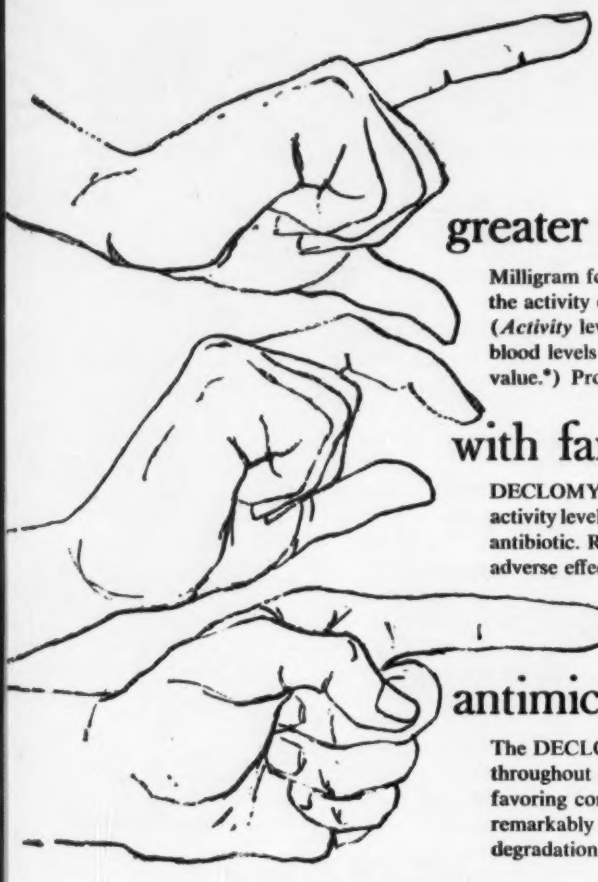

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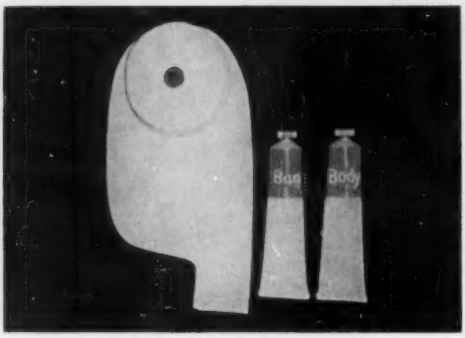

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EXPLORATORY FENESTRATION

of left humerus with partial removal by curettement of a lesion in the marrow cavity.

Radiographs, preoperative and postoperative. Color photographs, showing essential steps in the surgery.

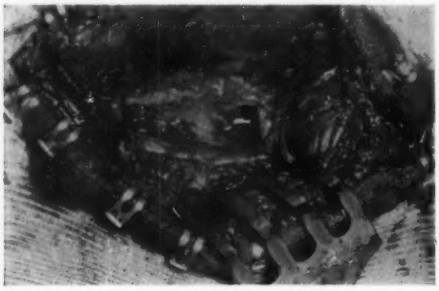
1. Incision at anterolateral aspect of the arm near insertion of the deltoid muscle.

(Turn page for additional illustrations)

Exploratory Fenestration (Continued)

The color photographs reproduced on this and the preceding page were made on Kodak Ektachrome roll film. 2 x 2 slides, also, were available to the surgeon for teaching and review, as well as black-and-white prints for files—definitive, fully objective materials *for use any time, anywhere!*

2. Small cortical window has been made in the humerus showing the underlying pathologic lesion.



3. Cortical window has been enlarged and the lesion in the marrow cavity partially curetted.

Note: Kodak Ektachrome roll film, used to make the illustrations for this picture sequence, may also be obtained for miniature cameras and sheet-film cameras.

4. Wound prior to closure. Two polyethylene tubes in place, one for suction, the other for insertion of antibiotics.

5. Technician preparing to radiograph patient in surgery to show extent of dissection in marrow cavity. Radiographs read wet, gave surgeon quick progress picture.

Postoperative radiograph made week following exploratory fenestration. Examination of resected tissue showed lesion to be a chondrosarcoma.

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Operative Ureteral Injuries*

WILLIAM E. FORSYTHE, M.D., and LESTER PERSKY, M.D., Cleveland

The incidence of operative ureteral injury varies, in reported series, between 0.5% and 3%. These usually occur as a result of surgical procedures, and the percentage when radical surgery is done may reach as high as 33%. Our exhibit is intended to outline the cause, symptomatology, and management of such trauma.

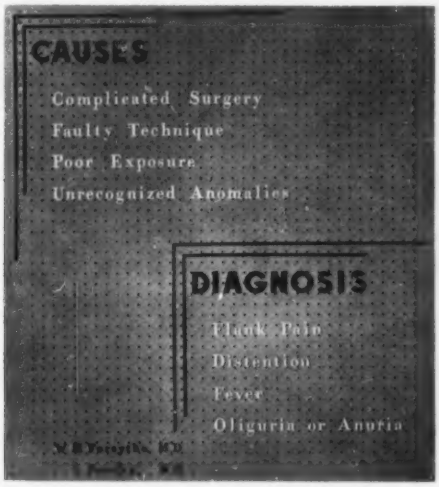


Fig. 1.—Outline of the causes and diagnostic features of ureteral injury. When flank pain or oliguria is present attempted cystoscopic passage of ureteral catheters is the first definitive step in elucidating the diagnosis.

Submitted for publication Aug. 11, 1959.

This exhibit was supported by the Kenneth Bliss Urological Fund.

From the Department of Surgery, Genito-Urinary Service, Western Reserve University and University Hospitals.

* Surgery Illustrated. Shown as a scientific exhibit of the Section on Urology at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

Fig. 2 (left).—Transection may be recognized at surgery by leakage of urine. Definitive treatment (illustrated below) can be done immediately.

Fig. 3 (right).—Ligation, a second type of ureteral injury, may be recognized immediately. At times deligation can be done in the immediate postoperative period.

Fig. 4.—Fistula formation may not be apparent immediately. It may be first discovered by the leakage of urine vaginally. It usually occurs secondary to a transfixing suture or ligature applied closely to the ureteral wall.

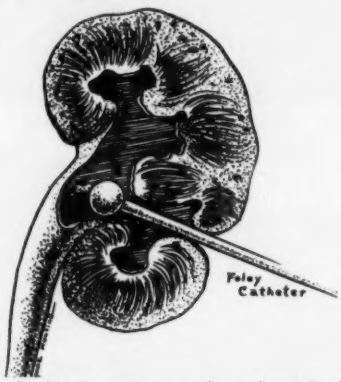


Fig. 5.—Nephrostomy may be and usually is the first step in definitive treatment. Its use protects the kidney and may at times be necessary bilaterally.

Fig. 6.—An indwelling ureteral catheter used as a splint facilitates primary anastomosis when transection is discovered.

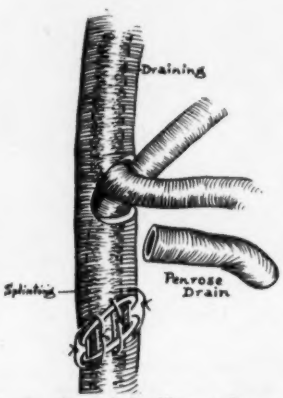


Fig. 7.—At times indwelling catheters directed above and below will splint an anastomotic area and direct the urine as well. With an extraperitoneal drain and the catheters brought out through a ureterotomy above, an excellent result should ensue.

Fig. 8.—An alternative to the splinting ureteral catheter or two catheters is the use of a T tube in the direct repair of transections.

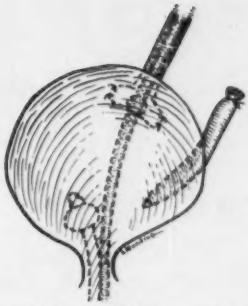


Fig. 9.—After preliminary nephrostomy and a short convalescence, when ureteral length is adequate at reexploration, a direct ureteroneocystostomy gives excellent results.

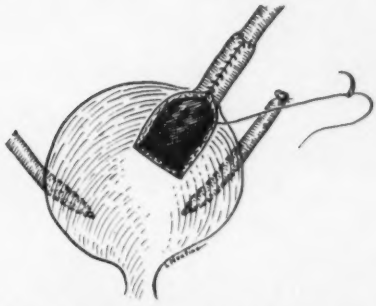


Fig. 10.—A technique to add length to the shortened ureter is the Ockerblad flap, which will usually bridge the deficit.

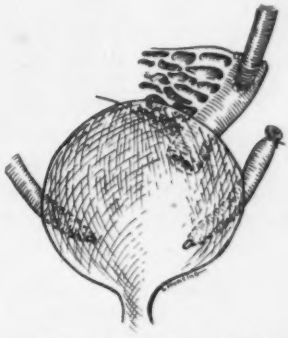


Fig. 11.—When the ureteral length is badly shortened the employment of an ileal graft will add needed length.

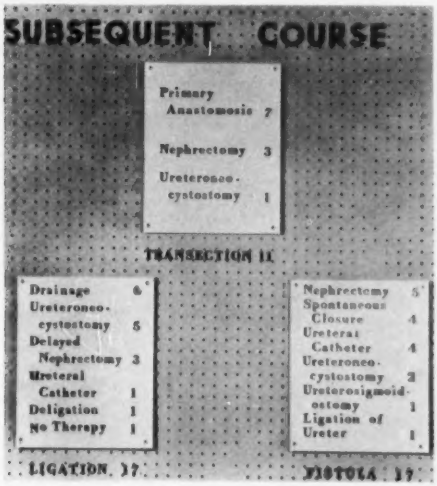


Fig. 12.—The total series included 45 cases. In 11 instances ultimate nephrectomy was necessary. Ligation of the ureter was done in one instance, when the patient's medical condition and attitude prohibited definitive repair.

Summary

Ureteral injury is usually iatrogenic. Its management often entails preliminary diversion by nephrostomy. Preliminary passage of ureteral catheters is the single measure which will enable the surgeon to identify the ureter prior to injury, and which will facilitate recognition of the injury when it occurs.

2065 Adelbert Road (6).

Strangulated Intestinal Obstruction*

Pathophysiology and Management

WILLIAM O. BARNETT, M.D., Jackson, Miss.

Is the Peritoneal Fluid of Late Strangulation Obstruction Toxic?

Fig. 1.—Animal exposed to fluid from strangulated bowel—dead at 24 hours.

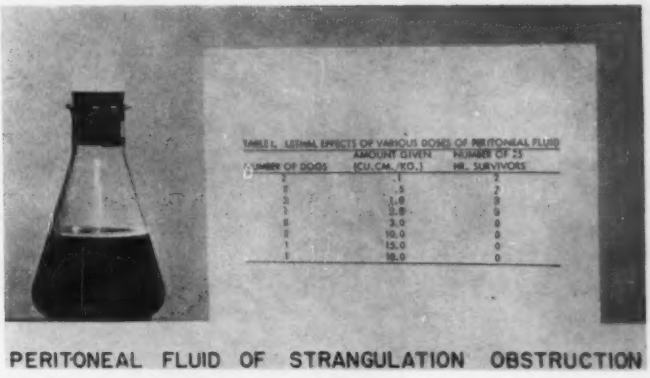
Fig. 2.—Exposure to fluid prevented by plastic bag—animal recovered after removal of gangrenous bowel.

Submitted for publication July 21, 1959.

From the Department of Surgery, University of Mississippi Medical Center.

* Surgery Illustrated. Shown as a scientific exhibit of the Section on Surgery, General and Abdominal, at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

Fig. 3.—Peritoneal fluid lethal in amounts as small as 2 cc. per kilogram.



Conclusion: The peritoneal fluid plays a major role in the lethal issue of strangulation obstruction.

What Component of the Fluid Is Responsible for Its Toxic Properties?

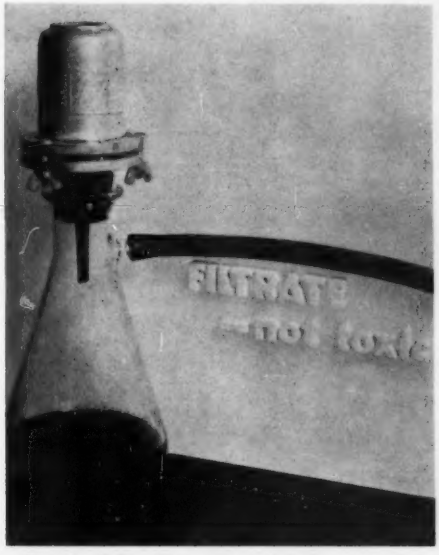


Fig. 4.—Seitz filter removed toxic properties—exotoxins not responsible for toxicity.

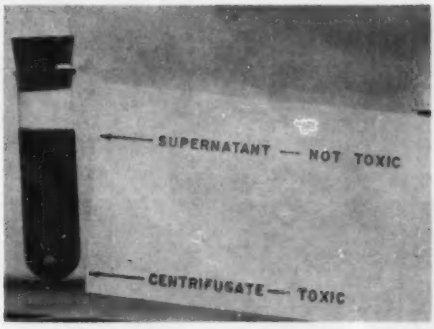


Fig. 5.—Centrifugation (17,000 rpm for 10 minutes) removes toxic portion—hemoglobin pigments not responsible for toxicity.

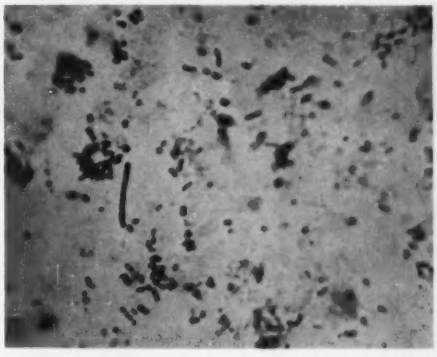


Fig. 6.—Smear of toxic centrifugate reveals many bacterial cells.

Conclusion: Bacterial cells appear to be responsible for the toxic properties of strangulation fluid.

After Peritoneal Exposure to Toxic Fluid Do Bacterial Cells Gain Access to the Blood Stream?

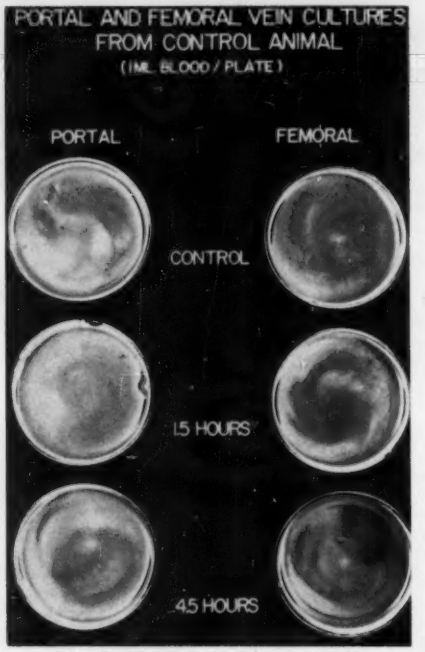


Fig. 7.—Control plates—no growth.

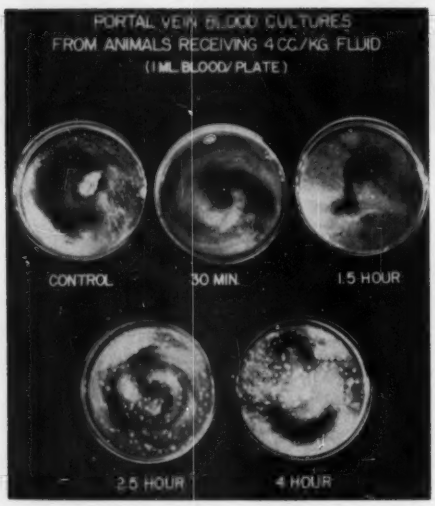


Fig. 8.—Portal blood reveals bacterial growth.

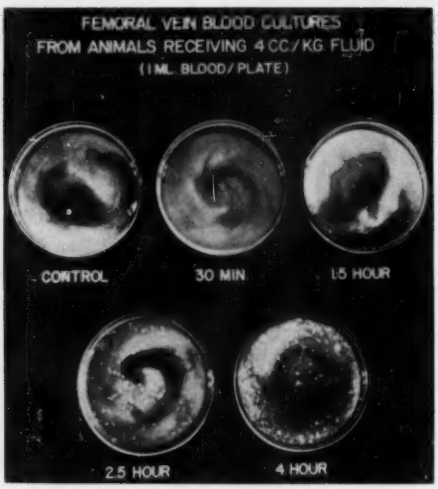


Fig. 9.—Femoral vein blood reveals bacterial growth.

Conclusion: Bacterial cells may be cultured from the portal or systemic blood after peritoneal exposure to fluid.

Will Antibiotics Prevent the Formation of Toxic Fluid?

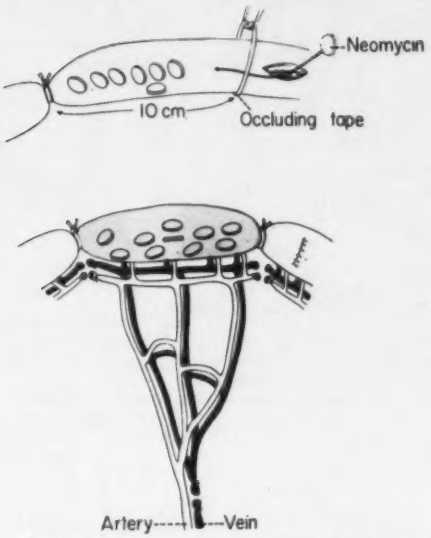


Fig. 10.—Neomycin eliminated lethal properties of fluid. Peritoneal fluid not toxic after 48 hours.

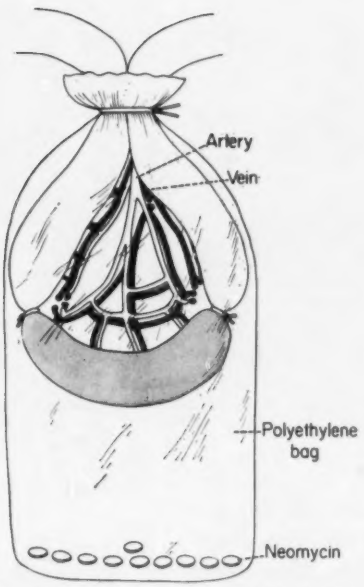


Fig. 11.—After 48 hours the fluid in the bag was not toxic.

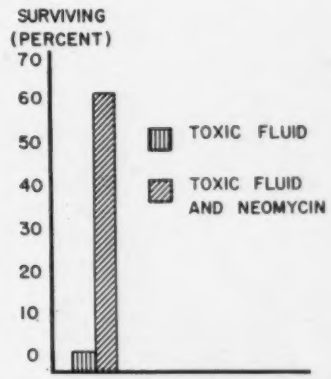


Fig. 12.—Comparison of survival rates in control animals and those treated with neomycin after intraperitoneal injection of strangulation obstruction fluid. Better survival rate in neomycin-treated dogs.

Conclusion: Antibiotics prevent the formation of toxic strangulation fluid.

Is It Possible to Immunize an Animal Against an Otherwise Lethal Dose of Strangulation Fluid?

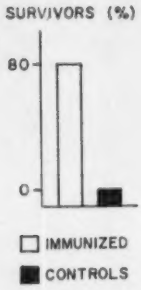


Fig. 13.—Ten normal dogs were given an intraperitoneal injection of toxic fluid (1 cc.) once a week for three weeks. These animals, along with 10 controls, were then given a intraperitoneal injection of fluid in the amount of 3 cc. per kilogram. The survival rate was better in the immunized group.

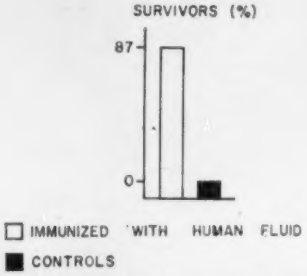


Fig. 14.—Eight normal dogs were given an intraperitoneal injection of fluid (1 cc.) once a week for three weeks. (This fluid was recovered from the peritoneal cavity of a *patient* with late strangulation obstruction.) These animals along with eight controls were exposed to the usual lethal dose of fluid. Previous injection with human fluid afforded protection.

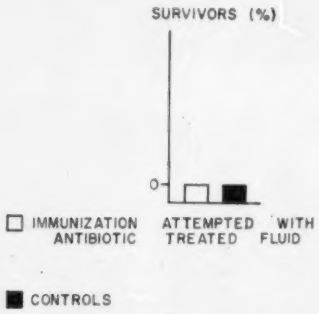


Fig. 15.—The normal dogs were given an intraperitoneal injection of fluid (1 cc.) each week for three doses. (This fluid was recovered after strangulation of a segment of ileum which had been treated with neomycin.) This chart shows the results obtained after exposure to toxic fluid (3 cc. per kilogram). Fluid without bacteria afforded no protection.

Conclusion: Previous injections of toxic animal and human strangulation fluid gave significant protection. Fluid arising from a neomycin-treated segment of bowel was not effective.

What Is the Effect of Toxic Strangulation Fluid upon Circulating White Blood Cells?

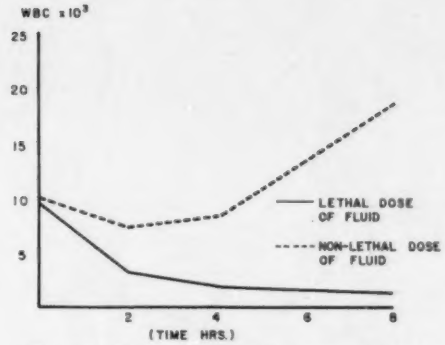


Fig. 16.—Mean white blood cell values following injection of lethal (3 cc. per kilogram) and nonlethal (0.2 cc. per kilogram) doses of strangulation fluid. Leukocytosis with nonlethal doses—leukopenia with lethal doses.

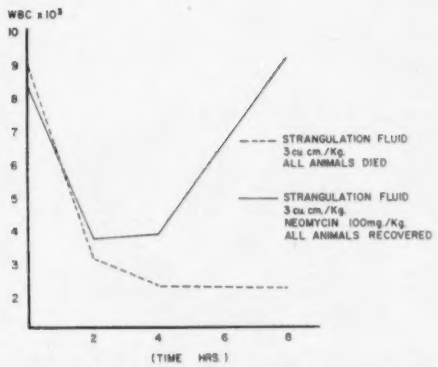


Fig. 17.—Comparison of mean white blood cell values following injection of fluid alone and in combination with neomycin. Antibiotics promoted leukocytosis.

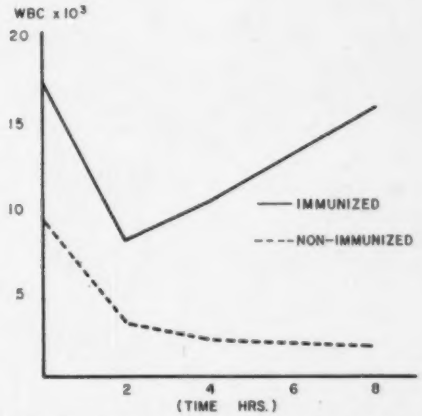


Fig. 18.—Comparison of white blood cell changes following injection of strangulation fluid (3 cc. per kilogram) into immunized and nonimmunized dogs. Immunized animals respond with leukocytosis.

Conclusion: Following exposure to toxic fluid, leukopenia indicate a fatal outcome, while leukocytosis is seen in those animals which survive.

Does the Intraperitoneal Injection of Strangulation Fluid Alter Body Fluid?

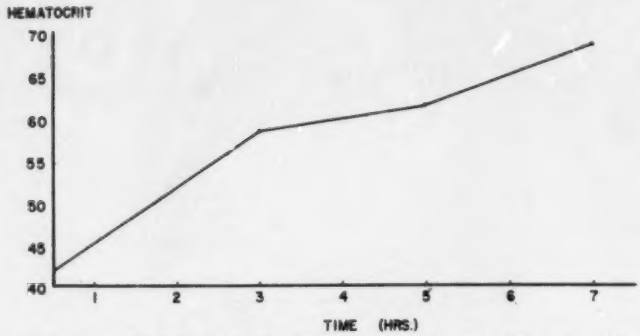


Fig. 19.—Hematocrit changes following intraperitoneal injection of fluid. Hemoconcentration follows exposure to fluid.

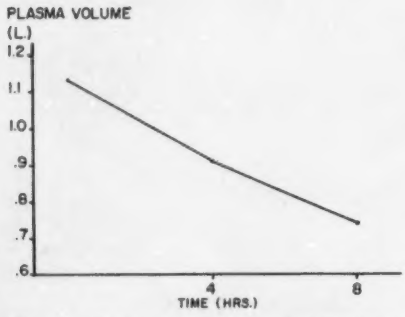


Fig. 20.—Plasma volume changes following exposure to strangulation fluid. Plasma volume decrease is progressive.

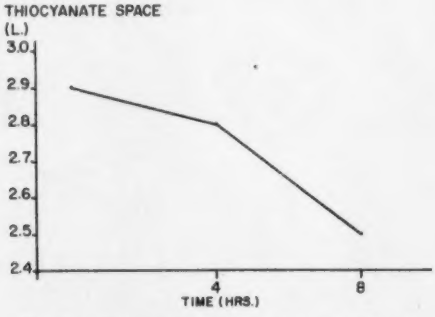


Fig. 21.—Thiocyanate space changes following exposure to strangulation fluid. Volume of the thiocyanate space is contracted.

Conclusion: Large amounts of fluid migrate to the peritoneal cavity after exposure to strangulation fluid.

What Is the Relative Toxicity of Peritoneal Fluid Resulting from Strangulation at Various Levels of the Gastrointestinal Tract?

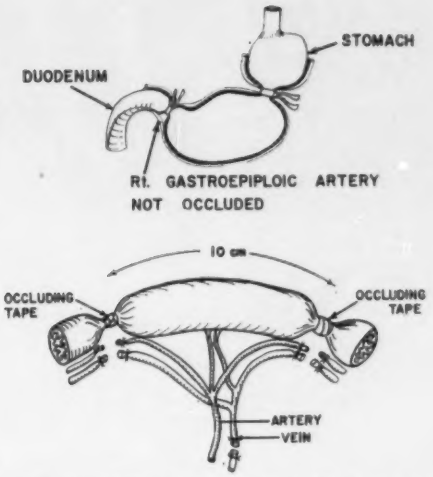


Fig. 22.—Method used to strangulate bowel of donor animals.

TABLE 1.—*Results in Donor Animals Following Strangulation of Stomach, Ileum, and Colon*

No. Dogs	Organ Strangulated	36-Hr. Survivors
4	Stomach	3
2	Ileum	0
2	Colon	0

TABLE 2.—*Results Following Injection of Peritoneal Fluid Resulting from Strangulation of Various Segments of the Gastrointestinal Tract*

Group	Organ Strangulated	Amount of Fluid Injected (Cc./Kg.)	No. Animals Injected	Survivors
1-A	Stomach	3	5	5
1-B	Stomach	3	5	0
	Stomach	2	5	3
2	Duodenum	3	5	4
3	Jejunum	3	5	3
4	Ileum	3	5	0
5	Colon	3	5	0
	Colon	2	5	0

Conclusion: The toxicity of peritoneal fluid resulting from strangulation increases from the upper to the lower gastrointestinal tract.

What Route of Antibiotic Administration Is Most Effective in Strangulation Obstruction?

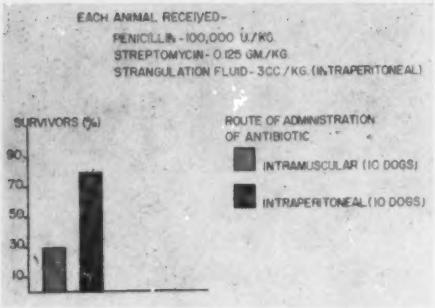


Fig. 23.—The efficacy of various routes of administration of penicillin-streptomycin in animals exposed to strangulation fluid. Pencillin was most effective after intraperitoneal injection.

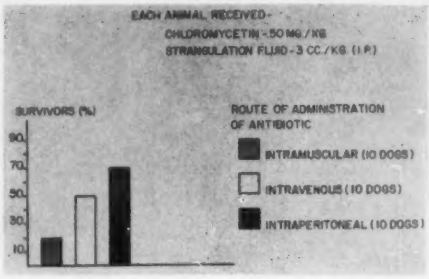


Fig. 24.—The efficacy of various routes of administration of chloromycetin in animals exposed to strangulation fluid. Chloromycetin gave best results after intraperitoneal injection.

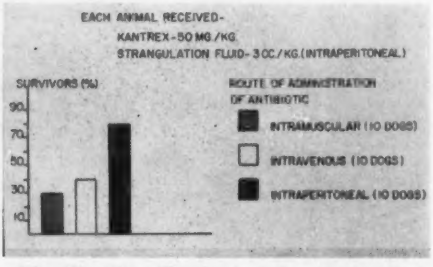


Fig. 25.—The efficacy of various routes of administration of kanamycin sulfate (Kantrex) in animals exposed to strangulation fluid. Kanamycin was most effective when injected into the peritoneal cavity.

Conclusion: The intraperitoneal route of administration was most effective for all antibiotics.

Does Delay in Administration of Antibiotics Affect the Survival Rate?

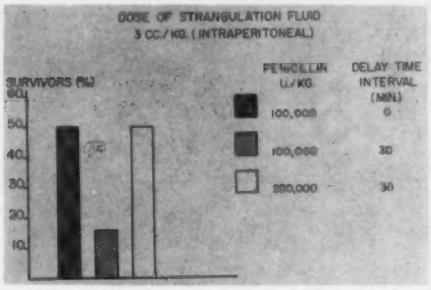
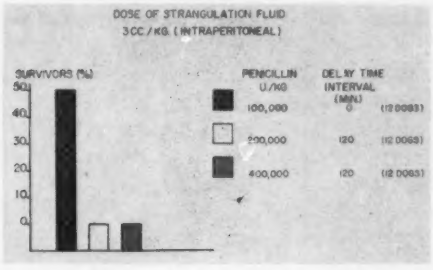


Fig. 26.—Results following delayed administration of penicillin—30 minutes. Results were improved by increasing the dose.



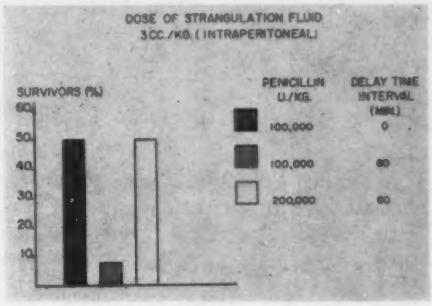


Fig. 27.—Results following delayed administration of penicillin—60 minutes. Results were again improved by increased dose of penicillin after delay of one hour.

Fig. 28.—Results following delayed administration of penicillin—120 minutes. After two hours delay, increased dose of penicillin was not effective.

Conclusion: Best results are obtained by early injection of antibiotics. After short periods of delay the survival rate can be improved by increasing the dose of antibiotics.

What Is the Best Method for Removing Toxic Fluid from the Peritoneal Cavity?

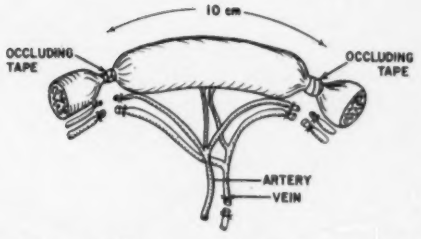


Fig. 29.—Method used to obtain strangulation fluid. Five normal dogs sustained strangulation obstruction of the ileum; 600 cc. of fluid was collected from the peritoneal cavities of these animals at or shortly before death.

Recipient Animals.— Twenty-eight normal dogs were given an intraperitoneal injection of fluid in the amount of 3 cc. per kilogram.

Group 1. The fluid was not removed.

Group 2. The fluid was aspirated from the peritoneal cavity after five minutes.

Group 3. After five minutes the fluid was aspirated. The peritoneal cavity was thoroughly irrigated with 1 liter of saline.

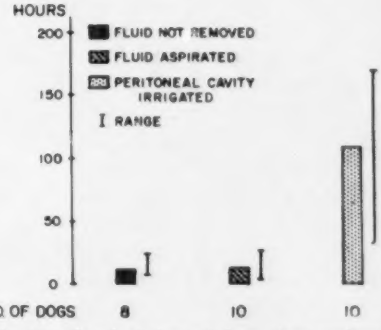


Fig. 30.—Survival time of dogs given peritoneal fluid. Survival time was longer following irrigation.

Conclusion: Thorough irrigation of the peritoneal cavity was the most effective method for removing fluid.

Some Details of Clinical Importance Which Have Been Adapted to Patient Management as a Result of These Studies

1. When the possibility of strangulation obstruction exists a peritoneal tap is carried out.

2. In a patient with intestinal obstruction the aspiration of dark, foul fluid strongly suggests strangulation.

3. One gram of kanamycin diluted in 200 cc. of saline is injected into the peritoneal cavity immediately.

4. The patient is carried to the operating room as soon as possible.

5. At laparotomy the strangulated bowel is withdrawn from the peritoneal cavity immediately.

6. Peritoneal fluid is then removed by suction.

7. The peritoneal cavity is irrigated with saline until it returns clear.

8. Resection of the gangrenous bowel is carried out, followed by anastomosis of normal bowel.

9. One gram of kanamycin dissolved in 200 cc. of saline is left in the abdominal cavity.

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Detailed Surgical Technique of Tracheal Fenestration, with a Report of Twenty-Six Cases*

E. E. ROCKEY, M.D., New York

Tracheal fenestration [1-5] is a new operative procedure for the treatment of chronic respiratory diseases. It is a skin-lined cervical tracheocutaneous communication equipped with two door-like skin valves at its external end. The valves can be opened at will for the purpose of tracheobronchial aspiration and medication, or self-aspiration and self-medication. When the valves are not held apart, the tracheocutaneous communication is airtight and leakproof; phonation and the cough mechanism remain normal. The purpose of this paper is to present the detailed surgical technique of tracheal fenestration, based on experience with 26 cases.

Operative Technique

A 4 in. long transverse skin incision is made over the lower anterior surface of the neck, about the level of the cricoid cartilage. This incision is then extended in such a way that one skin flap is outlined above, and one below, the level of the initial transverse incision. The base of each opposing skin flap is located approximately ½ in. from the midline, and its width is about 1 fingerbreadth (Fig. 1). The incision is deepened through the full thickness of the skin. In rare instances, the entire skin incision is deepened through the platysma also, in order to increase the mobility of the flaps. In most instances, through the initial transverse incision, the platysma is incised only to about ½ in. distal to the right and left of the midline (Fig. 2). (The skin flaps are never dissected from the underlying tissues.) The trachea is then exposed by splitting the strap muscles and, if needed, division of the thyroid isthmus (Fig. 3). A window is created on the exposed trachea by excising a 1.25 cm. length of two neighboring cartilages with the intercartilagenous tissue and the underlying tracheal mucosa (Figs. 4 and 5). The site of the window is at the level of the third and fourth tracheal rings, or lower if the total cervical tracheal length permits. The tracheal window edges are covered with skin. This is accomplished by inverting the distal margin of each of the two opposing skin flaps and fixing them to the tracheal mucosa with four interrupted 00 chromic sutures (Figs. 6, 7, and 12). In 14 (53.85%) of the cases reported here, 00 chromic catgut on a curved cutting needle was used for the inverting sutures. In the remaining 12 (46.15%) cases, placement of the inverting sutures required the use of special suture carriers and pickup forceps (Fig. 13). Description of the placement of the four "five step" inverting sutures is as follows:

Each suture is started near the base of the skin flap within the incisional wound. The needle pierces the subcutaneous tissue, platysma, and strap muscles (Step 1), then the 00 chromic suture is transferred from the curved cutting needle into a slightly

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From the New York Medical College-Metropolitan Medical Center, and St. Anthony's Hospital, Woodhaven, Long Island, N.Y.

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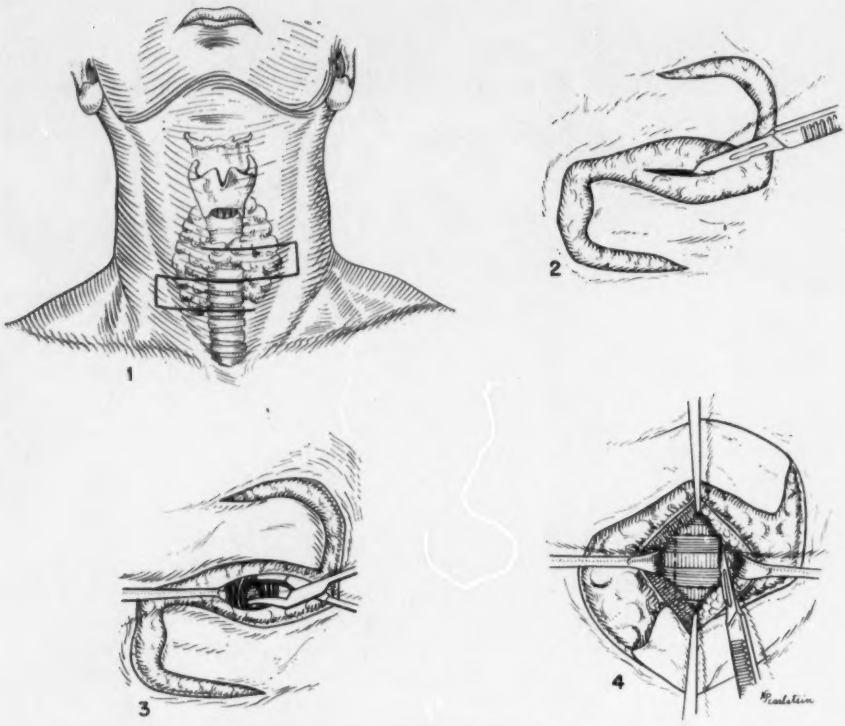


Fig. 1.—The initial incision is shown. Two opposing skin flaps are outlined. The base of the flaps is near the midline.

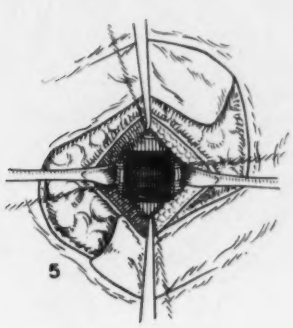
Fig. 2.—The skin incision is deepened and the platysma is incised in a transverse direction.

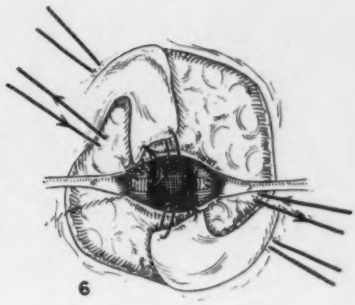
Fig. 3.—The trachea is exposed by splitting the strap muscles and, if necessary, the isthmus of the thyroid.

Fig. 4.—A window is outlined in the cervical trachea.

(*See paragraph* Attention *on page 888.*)

curved, special tracheal fenestration suture carrier. With the help of this suture carrier (Fig. 13), the inverting suture is advanced through the tracheal wall (Step 2), in on outward-inward direction, near the window edge (Fig. 12, *A*). The suture is then threaded again on the cutting needle and advanced through the skin flap near the junction of the distal margin and the same side margin of the skin flap where the suture was started (Step 3). This is performed in a subcutaneous tissue-to-skin direction. Then the same suture is threaded on an acutely curved special tracheal fenestration suture carrier, which is advanced through the tracheal wall in an inward-outward direction (Step 4), (Fig. 12, *B*). Finally, the suture is threaded again on the curved cutting needle and advanced through the strap muscles, platysma, and subcutaneous tissue (Step 5), bringing out the suture about 3-4 mm. distant from its starting point. Placement of the inverting sutures is much simpler when the trachea can be lifted upward and the initially used curved cutting needle utilized in carrying out all five suture steps. For maintenance of viability of the skin flaps, it is important that each of the four inverting sutures go to the appropriate window edge. The uppermost inverting suture goes to the upper window edge. The lowermost inverting su-





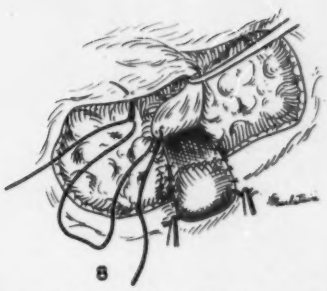


Fig. 5.—A 1.25 cm. length of two neighboring tracheal cartilages with the underlying mucosa is excised.
Fig. 6.—Four 00 chromic inverting sutures are in place.
Fig. 7.—The inverting sutures are tied; the distal margin of the skin flaps are inverted.
Fig. 8.—The skin valves are formed by doubling the outer two-thirds of the skin flaps. They are not dissected from the subcutaneous tissue.

(*See paragraph* Attention *on page 888.*)

ture goes to the lower window edge. The remaining two inverting sutures go to the right or left window edge, depending on which is nearer to their starting point.

The distal margin of each skin flap covers two of the four window edges (Fig. 7). The proximal two-thirds of each skin flap is doubled, without dissecting the skin from the subcutaneous tissue. The edges of the doubled part of the skin flaps are approximated with 000 interrupted Dermalon sutures (Fig. 8), creating the skin valves. The neighboring lateral edges of the already inverted distal two-thirds of the two skin flaps are approximated with two interrupted 000 Dermalon sutures on each side (Fig. 9), thus creating a skin tube. The remaining skin edges are approximated with 000 Dermalon sutures. By the time this is completed, a tight constricting ring is present at the base of each of the skin valves (Fig. 10, *a*). This is eliminated by a relaxing incision, approximately 1 cm. long, placed at a right angle to the constricting ring (Fig. 10, *b*). A clamp is inserted within the wound, and opened, in order to spread the skin edges.

Before the four inverting sutures are tied, a tracheotomy is performed below the level of the tracheal fenestration (Fig. 10). It is imperative to do it at this stage of the operation, to enable the surgeon to see

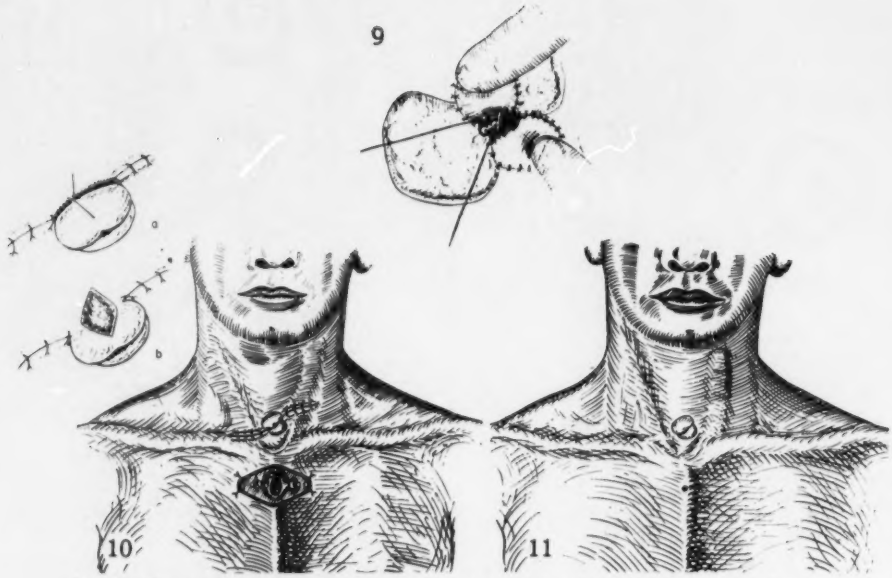


Fig. 9.—The skin tube is formed. The distal thirds of the skin flaps are approximated with two interrupted 000 Dermalon sutures on each side.

Fig. 10.—Tracheal fenestration is completed, and a mediastinal tracheotomy is performed below it.

Fig. 10.—(*a*) Outline of a 1 cm. long relaxing incision, placed half way over the basal area of the valve and half way over the neighboring skin. (*b*) The relaxing incision is widened, eliminating the constricting ring at the base of the valve.

Fig. 11.—The fenestration and site of the previous tracheotomy healed.

(*See paragraph* Attention *on page 888.*)

the site of the mediastinal trachea, where the cannula is inserted. Since at least two rings need to be left intact below the lower margin of the tracheal window, the tracheotomy cannula is inserted at the level of the seventh tracheal cartilage, or lower. A transverse skin incision is made, approximately 1 in. long, crossing the midline, about 1, 1½, or 2 fingerbreadths below the upper margin of the manubrium sterni. With the help of a Kelly clamp, through this incision a tunnel is created within the subcutaneous tissue, climbing over the manubrium sterni in order to reach the site of the tracheal opening in the mediastinum. After this tunnel is created, a skin hook is inserted into the mediastinal trachea at the site where the opening will be made. Then the trachea is pulled upward, the seventh or eighth cartilage is divided in a downward-upward direction. While the trachea is pulled upward, a No. 6 or 8 tracheotomy cannula, with its obturator in place, is inserted through the previously created subcutaneous tract. When this is accomplished, the obturator is exchanged with a trachotome (Fig. 14); its sharp tip is fed into the tracheal opening, and then the tracheotome, with the cannula threaded over it, is easily advanced into the tracheal lumen. The neck plate of the tracheotomy cannula is fixed to the skin with interrupted 00 silk sutures, and the tracheal fenestration is completed as described above (Fig. 10).

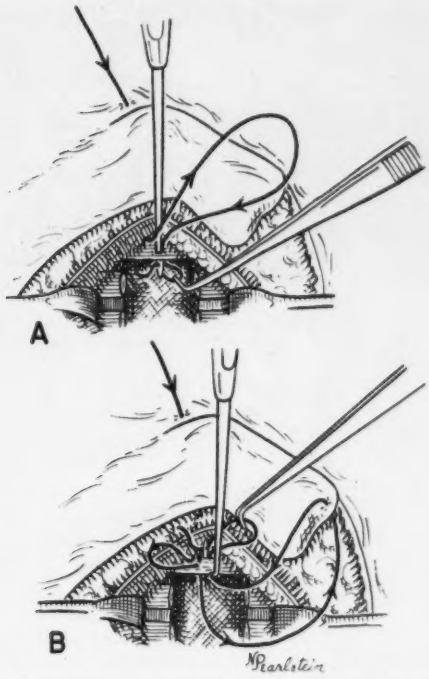


Fig. 12.—*A*, the second step in the placement of the "five-step" inverting suture: 00 chromic threaded into the slightly curved tracheal fenestration suture carrier is piercing the tracheal wall in an outward-inward direction. *B*, The fourth step in the placement of the "five-step" inverting suture: 00 chromic threaded into the acutely curved tracheal fenestration suture carrier is piercing the tracheal wall in an inward-outward direction.

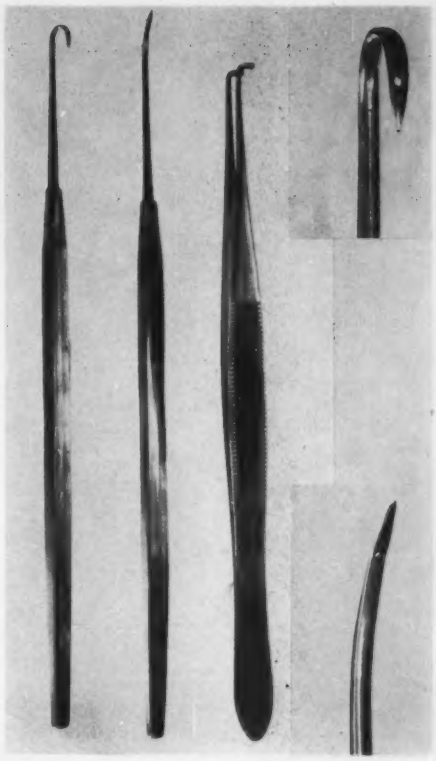


Fig. 13.—The slightly and acutely curved tracheal fenestration suture carriers. A right-angled tracheal fenestration pickup forceps.

Special Instruments Used for Operation and Equipment for Postoperative Care †

The equipment used for tracheal fenestration and postoperative care was as follows:

1. A slightly curved suture carrier (Fig. 13) to facilitate the second step in the insertion of the inverting sutures, namely, the piercing of the tracheal wall, near the window edge, in an outward-inward direction.

2. An acutely curved suture carrier (Fig. 13) to facilitate the fourth step in the insertion of the inverting sutures, namely, the piercing of the tracheal wall, near the window edge, in an inward-outward direction.

3. Right-angled pickup forceps (Fig. 13) to facilitate picking up the suture from the suture carriers, after they have already pierced the tracheal wall.

4. Mediastinal tracheotomy cannulas (Nos. 8, 6, 5, 4, and 3) (Fig. 14). These are extra long tracheotomy cannulas with the neckplate added at such an angle that it lies flat over the skin of the upper anterior chest wall after the insertion of the cannula.

5. Mediastinal tracheotomes (Nos. 8, 6, 5, 4, and 3) are obturators with their tip sharpened similarly to the tip of a trocar (Fig. 14). These tracheotomes facilitate the insertion of the cannula into the mediastinal trachea.

6. Selective bronchial suction catheters (Fig. 15).‡ They come in sets of six, ranging in size

† Suture carriers, pickup forceps, tracheotomy cannulas, and tracheotome (Items 1, 2, 3, 4, and 5) manufactured by George P. Pilling & Son Company, 3451 Walnut St., Philadelphia).

‡ Selective bronchial suction catheters manufactured by, and Detergicide distributed by, C. R. Bard, Inc., Summit, N.J. (Items 6 and 7).

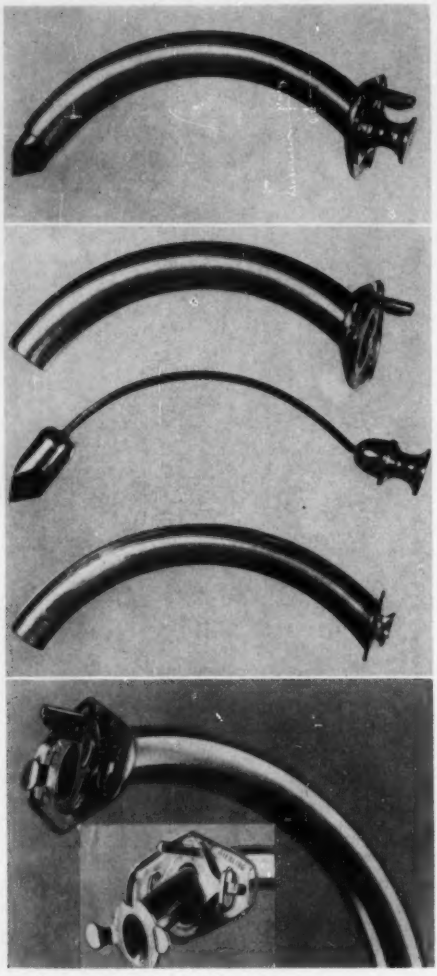


Fig. 14.—Mediastinal tracheotomy cannula with the neckplate attached at such an angle that it lies flat over the skin of the upper anterior chest wall. Mediastinal tracheotome, which is an obturator with its tip sharpened similarly to the tip of a trocar.

from F14 to F24. Because of their physical characteristics, they can be easily inserted into any designated lung or lobe. The smaller catheters also enter into segments or even subsegments of the designated lobe.[6]

7. Detergicide.‡ An aqueous solution of quaternary ammonium compound used for cold disinfection of the catheters (2.5 cc. in one gallon of tap water).

8. Tracheal fenestration sterilizing tray (Fig. 15).§ An acrylic plastic container with cover, for cold sterilization, designed to receive the catheters without curling them.

9. Tracheal fenestration serving or traveling tray (Fig. 15).§ An acrylic plastic container with cover and special inner springs to hold the catheters without curling them, while traveling. Also used as a serving tray during self-aspirations.

10. Tracheal fenestration suction unit (Fig. 16).‖ This is a small, (4×20×11 in.) and lightweight (10½ lb.), portable unit, which provides maximum suction power (27 in. of mercury).

Clinical Material and Results

From Jan. 30, 1956, to April 13, 1959, 26 patients were submitted to tracheal fenestration at Metropolitan Hospital, Flower and Fifth Avenue Hospitals, Manhattan General Hospital, Chest Pacilion, New York City, and St. Anthony's Hospital, Woodhaven, Long Island; 20 (76.92%) were white and 6 (23.08%) Negro; 21 (80.7%) were male and 5 (19.23%) female. Preoperative diagnoses were as follows: so-called "dry" emphysema, 7; advanced emphysema with chronic suppurative bronchitis, 14; bilateral, hopeless, far-advanced, active pulmonary tuberculosis, 2; active silicotuberculosis with advanced emphysema, 1; cystic fibrosis of the pancreas with advanced pulmonary suppuration, 2.

Clinical preoperative and postoperative data with end-results are listed in the Protocol (Table 1). The scope of this paper is to present primarily the details of operation, and of postoperative care. Deviation from the present operation and postoperative complications were as follows:

Four cases required a two-stage operation (Table 1, Cases 1, 4, 17, and 23).

Four cases required revision of the procedure (Table 1, Cases 8, 10, 12, and 19).

§ Tracheal fenestration sterilizing and serving trays manufactured by MO-AN Research Equipment Co., 62-05 84th St., Middle Village 79, N.Y. (Items 8 and 9).

‖ Tracheal fenestration suction unit manufactured by C. M. Sorensen Co., 50-19 47th Ave., Woodside 77, N.Y. (Item 10).

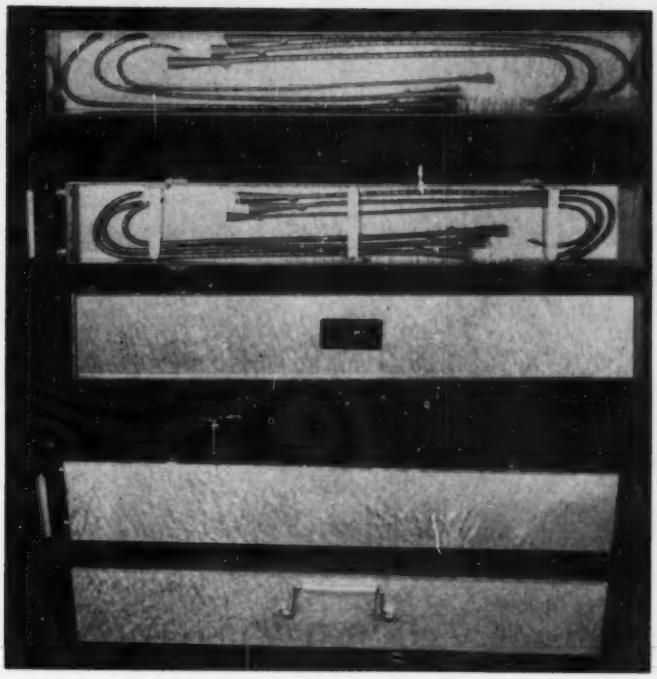


Fig. 15. — Tracheal fenestration sterilizing and serving trays with the selective bronchial suction catheters in place, with the covers off and with the covers on.

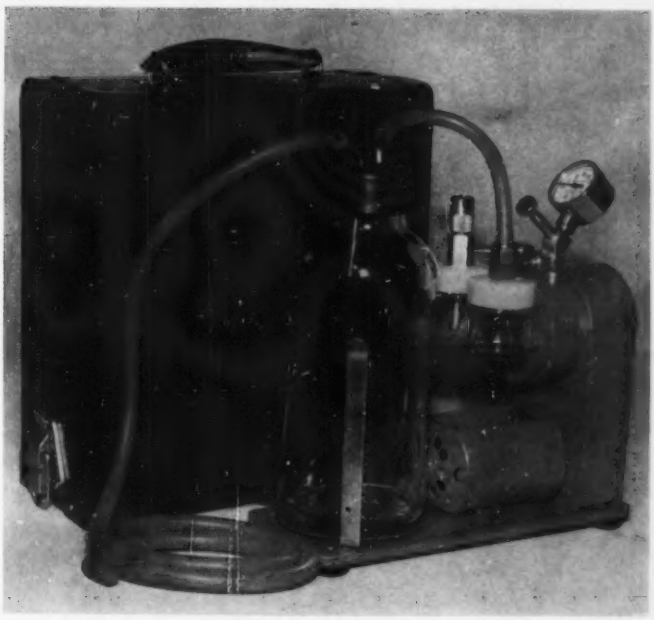


Fig. 16. — Tracheal fenestration suction unit.

TABLE 1.—*Protocol of Patients with Tracheal Fenestration*

Case No.	Color	Age, Yr.	Sex	Name of Hospital	Diagnosis	Date of Operation	Medical Supervision	Results
1	C	34	M	Met.	Bilat. far adv. active pulm. tbc.	1/30/56 4/ 5/56	Epstein	Palliation; died (4.5 mo.)
2	W	5½	M	F.F.A.H.	Cystic fibrosis	6/22/56	Slobody and Wasserman	Palliation; died (1.75 mo.)
3	C	32	M	Met.	Emphysema with infection	2/25/57	Epstein	Palliation; died (3 mo.)
4	W	52	M	St. Anthony's	Emphysema, "dry"	10/28/57 11/ 4/57	Blazsik	Marked rehabilitation
5	W	62	M	St. Anthony's	Emphysema, "dry"	12/ 2/57	Blazsik	Palliation; died (1.75 mo.)
6	C	63	M	Man. Genl.	Emphysema with infection	12/ 6/57	Edlin	Rehabilitation; died (8 mo.)
7	W	63	F	F.F.A.H.	Emphysema with infection	3/21/58	Schochet	Marked rehabilitation
8	W	61	M	Man. Genl.	Emphysema with infection	4/ 1/58 7/15/58	Snieska	Marked palliation
9	W	65	M	Met.	Emphysema, "dry"	4/15/58	Epstein	Palliation
10	W	67	M	St. Anthony's	Emphysema with active silicotuberculosis	4/23/58 5/29/58	Blazsik	Marked palliation; died (8.5 mo.)
11	C	65	M	F.F.A.H.	Emphysema, "dry" with cor pulmonale in failure	6/ 3/58	Boyd and Diamond	Palliation; died (1.75 mo.)
12	W	50	M	F.F.A.H.	Emphysema with infection and cor pulmonale in failure	6/ 5/58 11/ 6/58	Beck	Marked palliation; died (6 mo.)
13	W	82	M	St. Anthony's	Emphysema, "dry"	6/20/58	Blazsik	Marked rehabilitation
14	W	55	M	F.F.A.H.	Emphysema with infection and cor pulmonale in failure	6/23/58	Mayer and Rappaport	Marked palliation; died (2.5 mo.)
15	W	58	M	F.F.A.H.	Emphysema, "dry" and carcinoma of right lung	6/26/58	Mayer and Rappaport	Marked palliation; died (1.5 mo.)
16	W	53	M	F.F.A.H.	Emphysema, "dry" with arteriosclerotic heart disease (in and out of failure)	7/23/58	Gottesman	Marked palliation
17	W	62	M	St. Anthony's	Emphysema with infection	7/30/58 8/ 1/58	Blazsik	Rehabilitation; died (7.5 mo.)
18	W	56	M	St. Anthony's	Emphysema with infection and cor pulmonale in failure	9/18/58	Blazsik	Marked palliation
19	W	67	M	F.F.A.H.	Emphysema with infection	10/ 7/58 3/ 9/59	Noble	Palliation
20	W	37	F	F.F.A.H.	Emphysema with infection	10/16/58	Noble	Marked rehabilitation
21	W	65	M	St. Anthony's	Emphysema with infection	10/27/58	Blazsik	Rehabilitation
22	W	73	M	Met.	Emphysema with infection	11/ 3/58	Epstein	Rehabilitation; died (5 mo.)
23	W	55	F	F.F.A.H.	Emphysema with infection	1/21/59 1/30/59	Shub	Marked palliation; died (17 days)
24	C	33	F	St. Anthony's	Bilat. far adv. active pulm. tbc.	1/26/59	Blazsik	Palliation; died (1 mo.)
25	W	52	M	Met.	Emphysema with infection	4/ 6/59	Crispell and Stoff	Palliation
26	C	2½	F	Met.	Cystic fibrosis	4/13/59	Slobody and Wasserman	Palliation; died (2 days)

Inconsequential stitch abscesses developed in two cases (Table 1, Cases 17 and 25).

Minimal subcutaneous emphysema occurred during the first postoperative days in four cases (Table 1, Cases 5, 11, 14, and 23).

Minor adjustments for the purpose of bringing the skin valves nearer to each other were necessary in five cases (Table 1, Cases 6, 7, 9, 16, and 18). This consisted of a perpendicular skin incision about 1 in. long on each side of the fenestration, placed about ½ in. away from the external orifice of the skin tube. These incisions were closed by approximating the skin edges in a transverse direction[2] (Fig. 17). In two of these five cases, a triangular skin wedge was excised on each side of the fenestration, bringing the valves nearer.

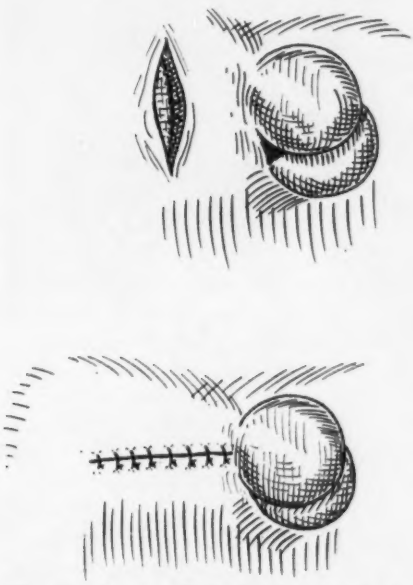


Fig. 17.—A perpendicular incision near the base of the valve, which, when closed transversely, brings the valves nearer each other and makes the fenestration more airtight.

Terminology Used in Clinical Evaluation

Palliation was accomplished when patients were made more comfortable than prior to operation. Exercise tolerance increased. Respiratory and pulse rate decreased. Color improved.

Marked palliation represents progressive improvement of pulmonary ventilation and exercise tolerance. Oxygen is no longer needed, and patients are discharged or ready for discharge from the hospital.

Rehabilitation was accomplished when ventilation and exercise tolerance increased sufficiently to enable patients to get around and attend to their personal needs without oxygen or the assistance of others. They are able to walk several blocks without respiratory difficulties.

Marked rehabilitation was accomplished when the condition of the patient improved sufficiently to allow a return to gainful occupation (when desired by the patient).

TABLE 2.—*Results*

	No. Cases
Palliation	10
Marked palliation	8
Rehabilitation	4
Marked rehabilitation	4
Total	26

Ten patients were palliated; eight gained marked palliation. Four were rehabilitated and four gained marked rehabilitation (Table 2). Of the 26 patients 11 are alive. Causes of death (Table 3) were as follows: bilateral, far-advanced pulmonary tubercu-

TABLE 3.—*Causes of Death*

	No. Cases
Bilateral far-advanced active pulmonary tuberculosis	3
Advanced emphysema and suppurative disease of the lungs	3
Cystic fibrosis of the pancreas with advanced suppurative disease of the lungs	1
Cerebrovascular accident	1
Carcinoma of the lung	1
Spontaneous tension pneumothorax	1
Hemorrhage due to duodenal ulcer	1
Coronary arterial occlusion	1
Pulmonary embolism from thrombophlebitis of leg vein	1
Subacute glomerulonephritis	1
Suicide	1
Total	15

losis, 3; advanced emphysema with chronic suppurative bronchitis, 3; cystic fibrosis of the pancreas with advanced suppurative disease of the lungs, 1; cerebrovascular accident, 1; carcinoma of the lung, 1; spontaneous tension pneumothorax, 1; hemorrhage from duodenal ulcer, 1; coronary arterial occlusion, 1; pulmonary embolism from thrombophlebitis of leg vein, 1; subacute glomerulonephritis, 1; suicide, 1.

Comment

Tracheal fenestration was well tolerated by all patients. Of the 26 cases, 22 were brought to the operating room in a Fowler position because of extreme dyspnea. After endotracheal intubation, tracheobronchial aspirations were performed from one-half to two hours before they were able to withstand a supine position. Large quantities of thick, tenacious, stringy material were aspirated from all of the cases. This includes seven patients with a history of no cough, or minimal cough, and no expectoration at all. These are the so-called "dry emphysema" cases. Patients spent three to four hours in the operating room. About 60% of this time was spent with tracheobronchial aspirations prior to, and during, operation. The extensive tracheobronchial toilet considerably increases the breathing capacity of these respiratory cripples, and for this reason they withstand the operation well. Operative mortality has been zero. By the time patients left the operating room, their color was better; their respiratory and pulse rate decreased, and they were able to lie flat on their backs. Tracheal fenestration is more of a hardship on the operating surgeon than on the patient. It is a complicated operation which can be mastered only if the surgeon has seen it performed at least once on a human being, and then practiced it on dogs before submitting patients to this procedure.

Tracheal fenestration should be performed in one stage, in order to obtain maximum functional result. Even cases with most advanced disease, who are markedly dyspneic, will withstand operation well, if extensive tracheobronchial aspiration is practiced before and during the procedure. It was not by choice that four cases in this series were submitted to a two-stage operation. The first attempt to perform tracheal fenestration (Case 1) made it clear that the dog operation had to be modified for human use.[3,4] This was accomplished by further animal experimentation, making the completion of the first operation possible, two months later, in a second stage. In Case 4, the skin flaps became detached from the trachea by the time the tracheotomy was completed. The difficulty arose from the fact that the site of the mediastinal trachea, where the tracheotomy cannula entered, could not be seen. In all subsequent cases, the tracheotomy was performed after the inverting sutures were placed, but before they were tied. This case was completed in a second stage one week after initial operation. Case 17 was operated on in two stages because of marked breathing difficulty developed by the patient by the time the tracheal window was created. This was due to the endotracheal tube inadvertently slipping into the right main bronchus. Though this was promptly corrected, the situation warranted completion of the operation in a second stage two days later. Case 23 was operated on in two stages because of faulty premedication. Contrary to rule that these cases are not to be given opiates at all, she received 50 mg. meperidine (Demerol) one hour prior to operation, and was unconscious when she reached the operating room. For this reason, only a tracheotomy was performed. The trachea was exposed through a transverse incision, placed at about the level of the cricoid cartilage. The tracheotomy cannula was placed exactly where the window would have been made. Nine days later, this opening was converted into the usual window and the opening for the tracheotomy cannula was placed below this level. Tracheotomies performed in this manner on cases with bulbar poliomyelitis would allow conversion of the tracheotomy to tracheal fenestration, in case

Fig. 18. — The appearance of the healed tracheal fenestration.

the neurological findings later prove irreversible. Of course, even this calculated and staged operation produces a second choice tracheal fenestration, as compared with the one-stage operation.

Four cases required revision of the tracheal fenestration. Case 8 had to be revised three and a half months after initial operation, because of excessive scar tissue formation at the junction of the skin flaps and the bronchial mucosa. The distal margin of the skin flaps (the part inverted into the trachea) did not remain viable. This area was repeatedly curetted; still the tracheal opening became sufficiently narrowed to make catheter insertion painful. Case 10 was a good fenestration except that inserted catheters went upward instead of downward. This was a special situation. This patient had no cervical trachea. His cervical air tract was made up of the thyroid and cricoid cartilages. The arch of his aorta was pulled upward. The exposed trachea between the upper margin of the aortic arch and the cricoid cartilage was located within the mediastinum. Because of this, at the time of initial operation, the tracheal window was created by the excision of a segment of the cricoid cartilage. Five weeks after the first operation, the tracheal window was placed at a lower level, leading to good function. Case 12 was taking prednisone (Meticorten) for over five years prior to tracheal fenestration. He continued taking this drug after operation. His wound healing was strikingly slow. In addition, under the skin, at the tracheal window edge, regenerated cartilage narrowed the tracheal window, making catheter insertions painful. Five months after initial operation, resection of the regenerated cartilage ring made the tracheal window larger and functionally satisfactory. Case 19 had a well-functioning tracheal fenestration while the patient practiced tracheobronchial aspirations, but he decided not to for two months. Subsequently, he could not pass the catheters any longer. Granulation tissue obliterated the tract at the junction of the skin tube and the trachea, and, five months later, the fenestration had to be revised.

The two cases with stitch abscesses and the three cases with postoperative subcutaneous emphysema were of no consequence. The stitch abscesses cleared after drainage, and the subcutaneous emphysema subsided after several stab wounds of the skin provided escape for air.

Postoperative Care

At the time operation is completed, the fenestration wound is covered with one layer of petrolatum gauze under dry dressing. The dressing is changed daily, with petrolatum gauze application only during the first three to four days. The sutures of the fenestration are removed in 12 to 14 days. Since patients start aspiration of the tracheobronchial tree on the third postoperative day, they might fumble over the area of fenestration and damage the operation if the sutures were removed before healing is completed. Sutures of the neck plate of the tracheotomy cannula are removed one week after operation, or even earlier, if the skin is tented and irritated by them. During the first 7 to 10 days after operation, tracheobronchial aspirations are carried out every 30 minutes, or oftener if needed. During the immediate postoperative period, when drainage is abundant, patients must not be left alone at any time until they prove able to aspirate themselves effectively. Within three to four weeks, the tracheal fenestration heals well enough to be used for catheter insertions. At this time, the tracheotomy cannula is removed and its site allowed to heal (Fig. 11). Appearance of the healed tracheal fenestrations (Fig. 18) shows only minor variations. Functionally, they comply with the criteria set forth, namely, they are airtight, leakproof, and do not interfere with phonation.

Present Indications

Tracheal fenestration is indicated in the following conditions:

1. Advanced cases of diffuse emphysema with or without clinically obvious secretion and with marked respiratory disability.

2. Excessive suppurative diseases (bronchiectasis) of the lungs when excisional surgery is contraindicated.

3. Fibrocystic disease of the pancreas with excessive pulmonary secretion.

4. Far-advanced cases of pulmonary tuberculosis for which no other form of therapy can be offered.

5. Bulbar type of poliomyelitis with irreversible neurological changes.

Summary

The detailed operative technique of tracheal fenestration is presented. Special instruments used for operation and equipment needed for postoperative care are described. Experiences with 26 cases are reported. This includes the immediate and longer term postoperative management, complications, and results. Of the first 24 cases, 8 (33.3%) with marked pulmonary insufficiencies were rehabilitated. The others have been improved to varying degrees. The operative mortality has been zero.

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525 Park Ave. (21).

ATTENTION

Revised drawings of Figures 1 through 11 were submitted too late to be published in this issue. They will appear in a later issue and will clarify points which now may not be clear to the surgeon.

Forearm Fractures*

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One hundred consecutive fractures of the forearm have been reviewed, which includes fifteen patients who had been referred to us and had been treated by one or more procedures elsewhere, which had either malunion or nonunion requiring an operative procedure at our institution. The purpose of this presentation is to illustrate the evolution of the methods of treatment of these fractures, evaluate the end-results, and record the conclusions. In 1950 we reported 320 forearm fractures.

The *prognosis* in young adults is considerably different from that in the older adults, and also, the open fractures, soft tissue injuries, and multiple fractures of one or both bones, and the site of the fracture tend to increase the period of treatment as well as influence the prognosis. No one method will suffice for all fractures of the forearm. Each must be individualized.

It is interesting to note in this series of fractures that the number of fractures occurring between 15 and 20 years and each decade thereafter up to 60 are almost identical in number, while there are only 10 fractures of both bones of the forearm in all the decades above 60. In other words, most of the fractures incorporated in this study were encountered in the active working period of life.

The radius was involved in 23 instances, the ulna in 13, Monteggia fracture in 6, radius and ulna in 58. Fractures of the lower third of the bone were encountered twice as often as those in the upper third, while the middle third fractures were almost equal to the sum of the other two with a predominance of both bone fractures occurring at the latter site.

There were 15 open fractures, 85 closed, and of these numbers 57 were the transverse or stable type fracture, while 43 of these were the comminuted or oblique unstable or irreducible type of fracture. This latter group are usually placed in a plaster cast as an attempt at closed reduction, but in eight of the unstable type we have operated initially.

There were 19 associated injuries to the soft tissues in this series, one in which the ulnar nerve was severed and one the radial nerve, the remainder being associated with dislocation of the radioulnar joint and general trauma of the forearm, including loss of skin, muscle, et cetera, which seemed to affect the ultimate outcome as far as the fracture was concerned, especially in the instances of severely compounded fractures which were denuded of their muscles, periosteum, et cetera. The severity of the local injury definitely influenced the healing of the fracture.

In 55 of the patients definitive treatment was carried out within 48 hours, 14 after 48 hours, while in 31 definitive treatment was not performed before one week and varying periods thereafter. The time element possibly plays an important role in healing, as reported by Smith in the *British Journal of Bone and Joint Surgery*, February, 1959.

In the entire series, 37 patients were treated in multiple stages, that is, had at-

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tempted closed reduction, followed by open reduction if we were unable to maintain position. Of the 37 patients requiring multiple-stage operations 3 were successfully treated by a secondary closed manipulation; 28 were operated upon in a secondary operation, while 3 required several procedures.

Comparing the success of open and closed treatment of fractures in all the patients in whom open reduction was carried out as a primary treatment, including the later as well as the open fractures, we find that there were 15 open fractures and 8 closed which were treated by immediate surgery. Of these, 13 required further treatment. In the 77 closed reductions, 56 healed without further complication, 21 requiring further treatment.

In regard to the methods of treatment by open reduction, early in our series 15 fractures were treated by plate, 9 primarily and 6 secondarily; 4 had been plated elsewhere; 14 of those 15 required further treatment, that is, bone grafting. The remainder of the cases were treated by intramedullary fixation of one type or another, either Steinmann pins or Rush pins. When this procedure was used, substituting either immediate grafting or onlay grafts using cancellous matchsticks around the fracture site, or onlay type of graft for stability and for the replacement of bone loss, the chance of success in this series was 84%.

TABLE 1.—*Types of Operations*

	Primary	Secondary
Plate and screws	9	6
Intramedullary fixation	5	6
Graft and screws	2	5
Intramedullary fixation and grafts	7	20
	23	37

9 additional operations for excision of radial and ulnar heads, tendon transplants, etc.

The follow-up period in these fractures was 42 days to 120 months, the average being 612 days. The 42-day patient was lost track of, hence the short period of follow-up. This long period of follow-up can be explained by the method employed in the Fracture Clinic and the complicated problems.

TABLE 2.—*Success of Various Operations*

	Successful	Unsuccessful
Plate and screws	1	14
Intramedullary fixation alone	8	3
Grafts and screws	2	5
Intramedullary fixation and grafts	24	3
	35	25

84% successful with intramedullary pin and/or graft

Length of follow-up	
Minimum	42 days
Maximum	120 months
Average	612 days

Average length of immobilization	Internal	External
Minimum	28	35
Maximum	330	420
Average	146	98

Infection	Closed Fracture	Open Fracture
Previous to admission	0	0
Acquired here	3	4
Poor results among those 7	5	

This points out importance of infection in the result

Complications

Four cases developed nonunion which was persistent. Of these four, one had a persistent infection; one had repeated grafting of the ulnar fracture, which eventually did not heal, but the patient developed a good functioning forearm. In four instances, the internal fixation was removed, owing to pin irritation at the wrist or for some other cause, from what was thought to be satisfactorily healed bones. It was found that subsequent bowing of the fracture site occurred, necessitating further periods of external fixation. The lesson learned from these four patients has taught us to maintain the internal fixation for a much longer period of time than perhaps x-ray evidence would indicate. It is for this reason that we have used the Rush nail, which is left in for several weeks or months

after all external fixation has been removed. One complication that was encountered in this series was a shearing of the extensor pollicis longus by the hook of the Rush pin.

The average period of internal fixation in this series has been 146 days. The average period of external fixation was 98 days. In all instances, we have used external fixation combined with the internal fixation. In all, there were seven cases of persistent infection which recurred following two simple fractures in which open reduction was used, while five of the infections were in open fractures of varying degrees of severity. Five poor results were obtained from this group, showing the poor prognosis of fractures complicated by infection.

Results

In evaluating our results, we accept as good results or satisfactory results those cases which had no more than 15 degrees of limitation of extension of the elbow, not more than 60 degrees of limitation of pronation and supination. Thus classifying our end-results, it was found that we had 15 unsatisfactory results from the physician's standpoint, but from the patient's standpoint there were 12 instances in which the result was unsatisfactory. If the forearm was in good functional position and the patient had a good elbow and shoulder, he was able to compensate well even though he had lost some pronation and supination. The degree of loss was somewhat in proportion to the extensiveness of the injury, especially those affecting soft tissues, and the multiplicity of procedures necessitating prolonged immobilization of the part. There was no instance of Volkmann's contracture, although there were some instances in which certain muscles were badly damaged at the time of the injury, thus influencing the function of the hand and wrist. In three of these patients, tendon transplants to promote better function, particularly in extension of the wrist and fingers, were performed.

In reviewing the last 37 cases in which open reduction of the fracture was performed with internal fixation, we found that we have used intramedullary fixation alone in 8 cases, internal fixation with intramedullary pins and screws with an onlay graft in 9 patients, internal fixation with matchstick or cancellous bone grafts in 20 instances. We also feel that our improved results obtained in these later cases have been due to the fact that we have been following the premise of the experimental work by McNaab and by Trueta in which they have definitely shown that stripping of the periosteum deprives the fractured ends of the bones of circulation; hence in our surgery we have done very little except to approximate the ends of the bone, and, if there was an area sufficiently exposed for grafting, cancellous bone was applied in the form of matchsticks around the fracture site. We believe that once the decision to operate is made, delay is not very important, but what is done is. At best these are difficult fractures to care for, hence the initial surgery is most important. For all *secondary open operative* procedures, it was our feeling that autogenous bone, and usually iliac bone, was the graft of choice, and these were used particularly for major replacements for bone loss, butterfly-type fractures, et cetera. Frozen bank bone was used in the fractures treated primarily by open reduction.

The pins that are used should fit firmly in the intramedullary canal of the radius and ulna so as to hold the bones in firm fixation, and a small wire such as Kirschner wires or small Steinmann pins should not be used except in an unusual case where the intramedullary canal is so small that other types of fixation cannot be used.

In conclusion, we feel that all open fractures should be fixed by intramedullary pin if possible, all soft tissue loss be corrected and denuded areas grafted, and that any open reduction performed on these difficult fractures should be accompanied by primary bone graft when possible, either the

matchstick type or the onlay graft if indicated. In midshaft and both bone fractures we have used the intramedullary pin on one bone and graft on the other in several instances. In late secondary operations these patients should have the benefit of the very best type of graft possible, that being an autogenous graft and preferably good cancellous bone such as the ilium provides. We feel that internal fixation should be left in situ for many weeks after the external fixation has been removed and the arm has been in full use, and, as a rule, pins are not removed for several months unless they are causing some irritation to the surrounding tissues. Results: although some patients required multiple procedures and five had poor results, and included are patients with malunion, bone loss, and open fractures as they presented themselves to our clinic, the over-all results of the methods presented have seemed to justify their use. We have not used plates except in unusual situations. Most late patients referred to us with complications are those in whom bone plates have been applied primarily. Examples are shown.

Don'ts

1. Don't use too small intramedullary pins or wires.
2. Don't use inadequate fixation of any kind.
3. Don't remove pins too soon.
4. Don't wait to treat improper alignment or unstable fractures of one or both bones of the forearm.

Conclusions

1. One attempt at closed reduction and casting in all adult forearm fractures is justified.
2. For unstable fractures, open reduction with intramedullary fixation and immediate grafting, either barrel-stave, matchstick, or, when necessary, onlay grafts, is recommended. Casts are used routinely.
3. Inadequate fixation and infections cause all major complications.

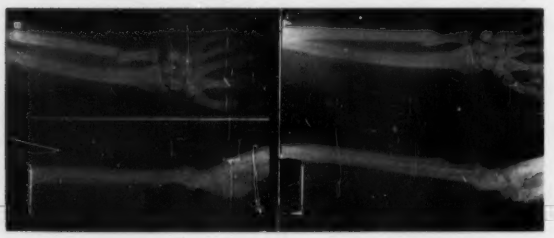


Fig. 1.—Fracture of ulna treated by closed reduction and cast. Some displacement accepted with a good functional result.

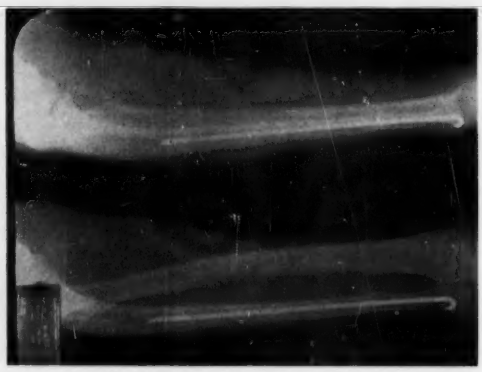


Fig. 2.—Intramedullary pin for an unstable fracture of the distal third of ulna.

Fig. 3.—Intramedullary pin used to hold an oblique unstable fracture of the radius.

Fig. 4.—Closed reduction of the distal radius with rotation of fragments. Displacement occurs in this type of fracture because of pronator quadratus contracture, and open reduction is required.

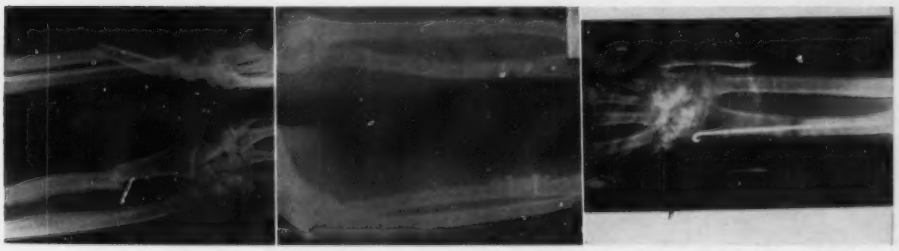


Fig. 5.—Open reduction is usually necessary in this fracture. This may be accomplished with an onlay bone graft or an intramedullary pin.

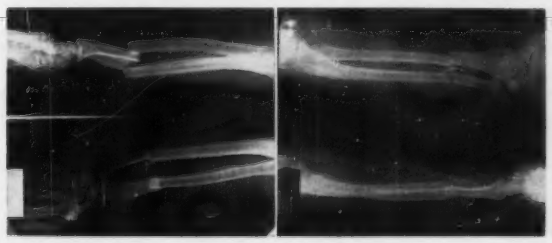


Fig. 6.—Fractures of both bones of the forearm, if transverse, may be treated by closed manipulation. Periodic x-rays are necessary because of frequent displacement.

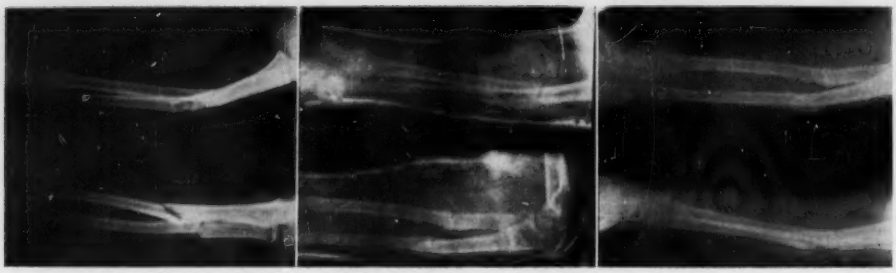
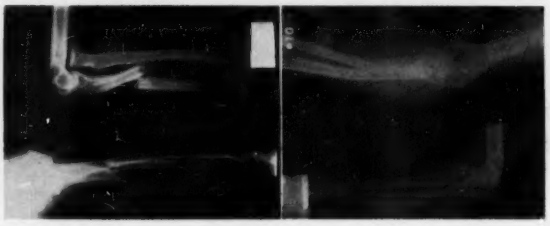


Fig. 7.—Oblique undisplacement fracture of both bones of upper middle third treated by closed reduction. Healed with slight angulation but excellent function.

Fig. 8.—Monteggia's fracture treated by closed manipulation (supination and flexion of elbow). Excellent functional result.



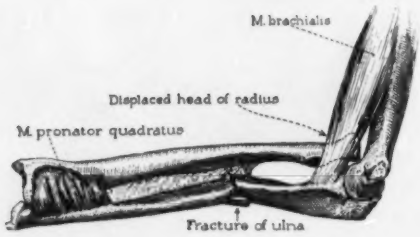


Fig. 9.—Monteggia's fracture. The result of a direct blow fracturing the upper middle third of the ulna and dislocating the head of the radius.

Fig. 10.—Open reduction and intramedullary fixation of the ulna with excision of the radial head (late). Frequently it is possible to replace the radial head and repair the joint capsule.

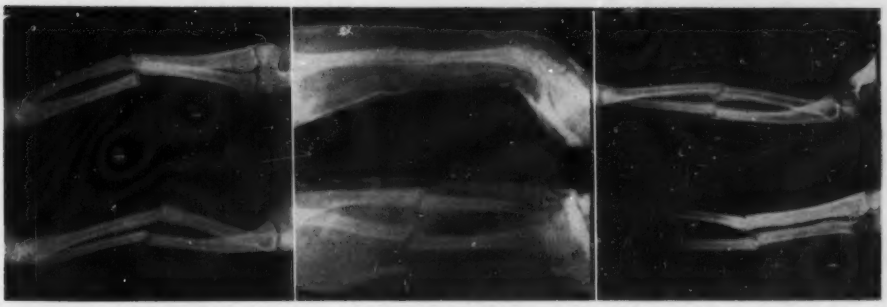


Fig. 11.—Transverse fracture of both bones of forearm. Apposition still present. Closed reduction resulted in union.

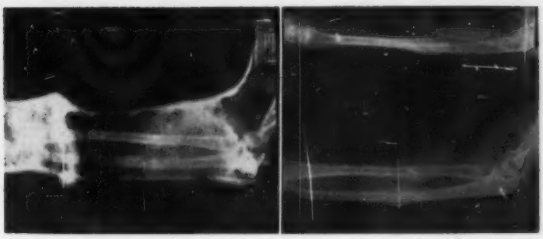


Fig. 12.—Oblique fractures (unstable) of both bones of forearm treated by transverse Kirschner wire fixation and casting.

Fig. 13.—Fracture of both bones of forearm with overriding treated by open reduction with intramedullary fixation of ulna and onlay grafting of radius.

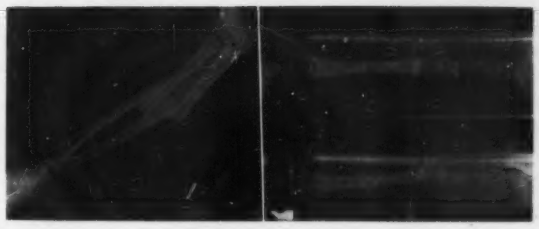


Fig. 14.—Fracture of both bones of forearm treated first by closed reduction. Overriding still present. Open reduction with intramedullary fixation and bone grafting. (Note: Small intramedullary canals necessitated small flexible pins.)

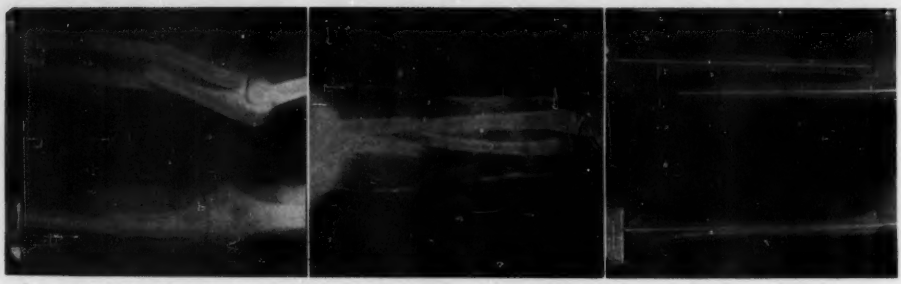
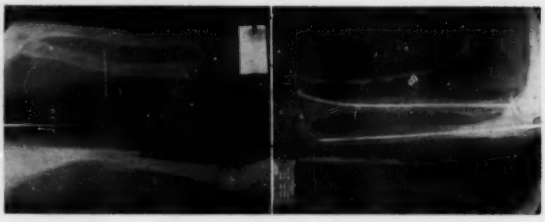


Fig. 15.—Fracture of both bones of forearm treated initially by manipulation. The ulna is still displaced. Open reduction with intramedullary fixation and bone grafting.

Fig. 16.—Comminuted fracture of middle third of both bones of forearm. Open reduction with intramedullary fixation and bone grafting (Note: all fragments retained.)



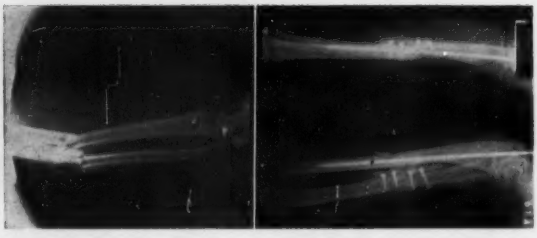


Fig. 17.—Fracture of both bones of the proximal third of forearm. Open reduction with intramedullary fixation of ulna and onlay bone graft of radius.

Fig. 18.—Oblique fracture of both bones of forearm, an unstable fracture treated initially by closed reduction. Open reduction with intramedullary fixation of ulna and onlay bone graft of radius.

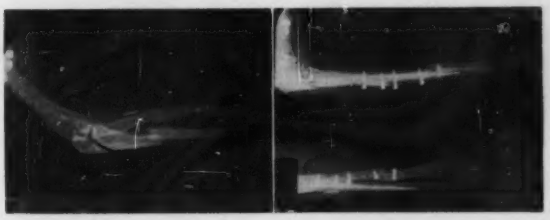


Fig. 19.—Multiple fracture of both bones of forearm. Intramedullary pin and double onlay bone grafts.

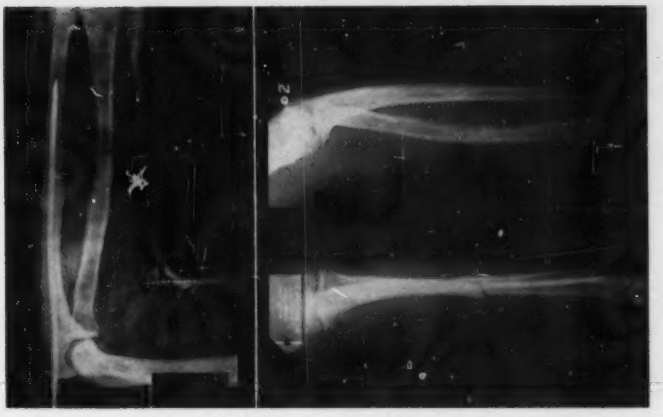


Fig. 20. — Steinmann pin holding radial fragments separated. Removal of pin and barrel stave grafts on radius.

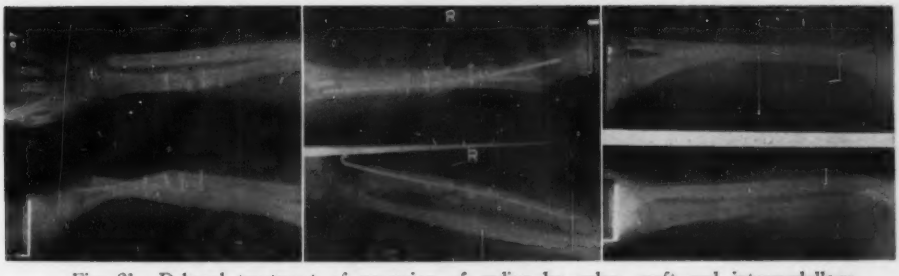


Fig. 21.—Delayed treatment of nonunion of radius by onlay graft and intramedullary fixation. Angulation and slow healing following too early removal of intramedullary pin.

Fig. 22. — (Left). Open fracture. Bone loss and inadequate wire fixation—nonunion. (Right). Open reduction—intramedullary Rush nail and autogenous iliac onlay graft.

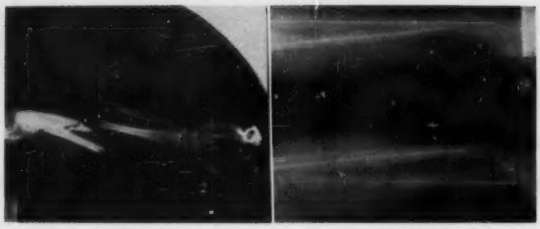


Fig. 23.—Open severly comminuted fracture. Intramedullary fixation—immediate.

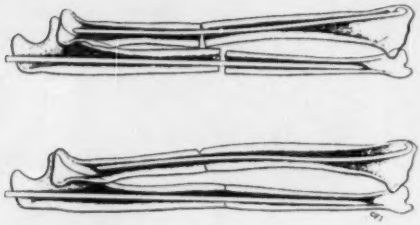


Fig. 24.—Illustrating the effect of the intramedullary pin on long curved bones.

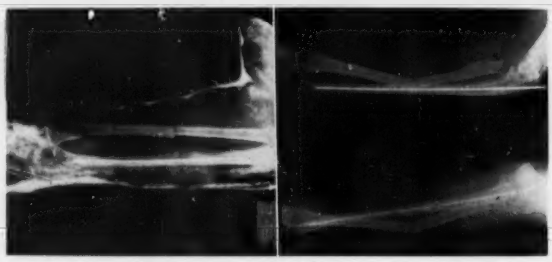


Fig. 25.—Intramedullary pin to ulna with resulting rotation of radial fragments. Malunion of radius.

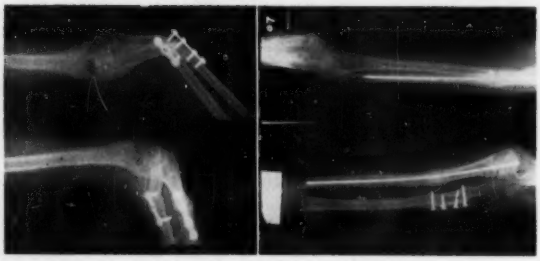


Fig. 26.—Malunion and nonunion following plating. Intramedullary nailing and grafting of ulna and onlay graft of radius.

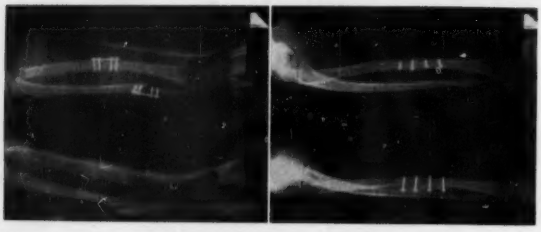


Fig. 27.—Plating of both bones with nonunion. Onlay graft to radius; peg graft in ulna.

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Tumors of the Parotid Gland*

Their Surgical Management

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Mixed Tumors of the Parotid Gland

The term "mixed tumor" refers to the commonest group of neoplasms of the salivary glands. In the parotid gland, they make up approximately 80% of the benign tumors. They are slowly growing, firm, encapsulated, and freely movable lesions, varying from 1 to 15 cm. in diameter. Grossly, the primary tumor occurs as a single nodule whose cut surface may show varying degrees of cartilaginous consistency. Recurrent lesions are usually multicentric. Long-standing mixed tumors may undergo malignant transformation, as shown elsewhere in this exhibit.

Fig. 1.—Recurrent mixed tumor. Original tumor was removed by excision.

Fig. 2.—Recurrent mixed tumors are multinodular; primary lesions are unilocular.

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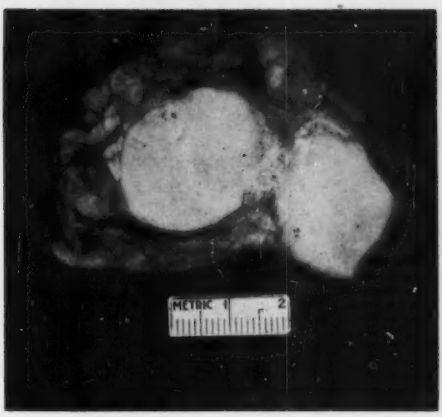
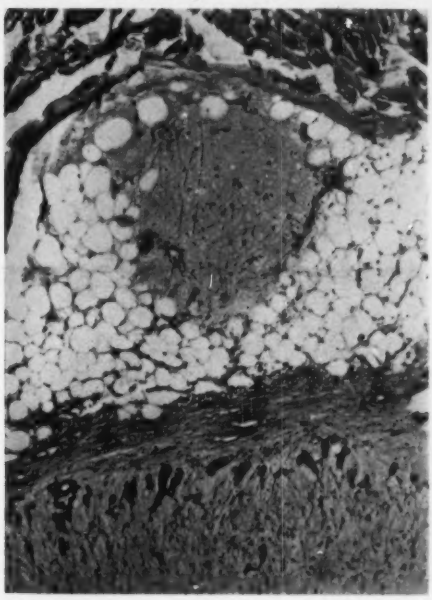


Fig. 3.—Mixed tumor. Typical encapsulated solitary nodule showing grayish-white or yellowish cut surface. Cartilaginous areas appear semitranslucent.

Fig. 4.—Mixed tumor. This is the classic histologic appearance, demonstrating islands of epithelium merging with cartilaginous myxoid elements.

Fig. 5.—Mixed tumor. This mixed tumor has extended outside the tumor capsule. Such excrescences or imperfect encapsulation account for failure of enucleation procedures.



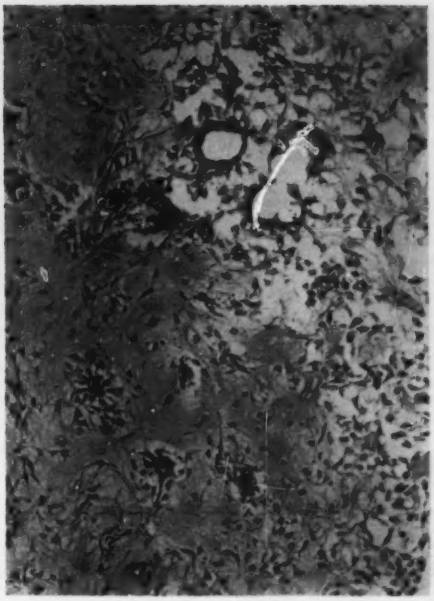


Figure 4

Fig. 6.—Mixed tumor. This recurrent mixed tumor shows multiple nodules scattered throughout the substance of the gland.

Less Common Benign Tumors and Cysts of the Parotid Gland

1. Warthin's tumor (papillary cystadenoma lymphomatosum) makes up about 7% of the benign group. It occurs predominantly in male patients and is located typically at the posterior margin of the lower pole of the gland. In about 10% of cases, the lesion is bilateral or multicentric. Grossly, these tumors are encapsulated, soft and cystic. 2. Of almost equal frequency are retention cysts of ductal origin. 3. Oncocytic adenomas (oncocytomas) make up about 1% of parotid tumors. 4. Occasionally, an inflammatory lymph node, a hemangioma or a keratoma occurs within the substance of the parotid.

Fig. 7.—Hemolymphangioma involving and almost totally replacing the parotid gland.

Fig. 8.—Hemangioma of parotid gland partially involving the external ear.

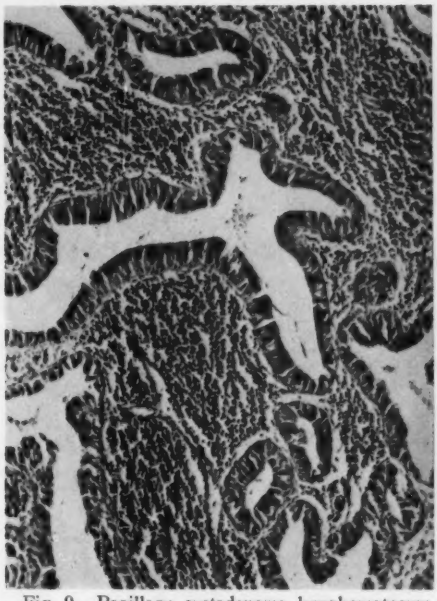


Fig. 9.—Papillary cystadenoma lymphomatosum. Acidophilic epithelial cells are present in a lymphoid matrix. Note cells around glandular or cystic spaces with papillary infolding.

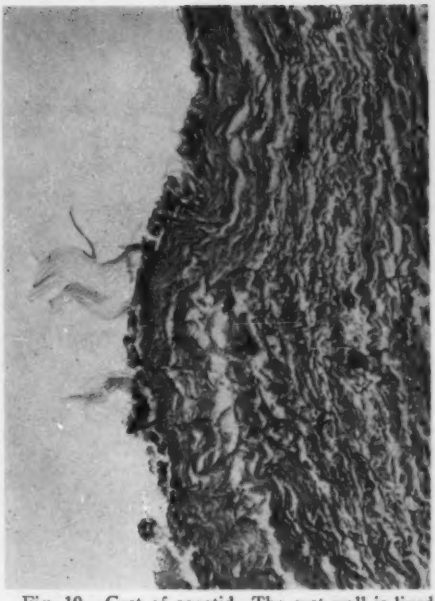


Fig. 10.—Cyst of parotid. The cyst wall is lined by cuboidal or columnar epithelial cells with slight production of mucus.

Fig. 11.—Oncocytic adenoma. The tumor is composed of large cells with voluminous acidophilic and granular cytoplasm. The nuclei are small and regular, and mitotic figures are absent.

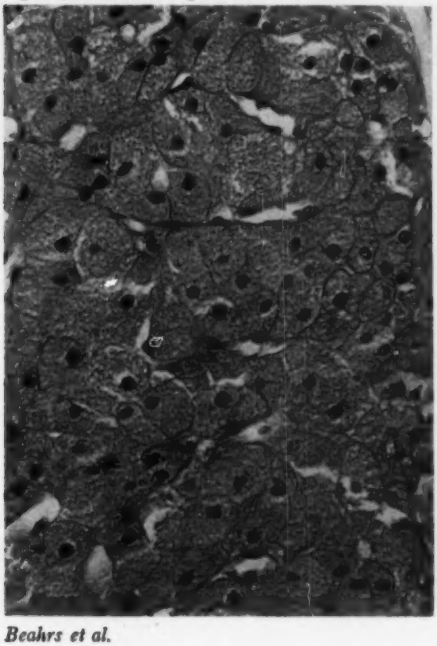
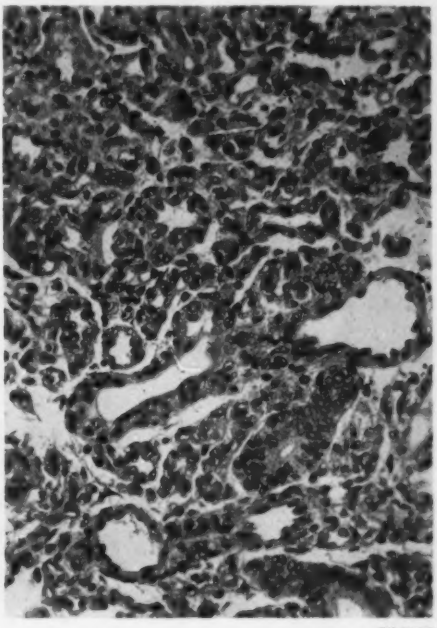


Fig. 12.—Hemangioma. The parotid tissue is largely replaced by proliferating endothelial cells forming small capillary spaces.



Inflammatory Lesions of the Parotid Gland

1. Mikulicz's disease (benign lymphoepithelial lesions) is a rare condition involving one or more of the salivary glands. It is seen more frequently in women than in men. The gland is diffusely enlarged and rubbery. 2. Boeck's sarcoid (uveoparotid fever) also may involve one or multiple salivary glands as a firm, nontender swelling. 3. Chronic sialadenitis is a painful, fluctuating, diffuse swelling of one or occasionally both parotid glands. The inflammatory reaction is a result of cicatricial or calculous obstruction of the ducts.

Fig. 13.—Bilateral recurrent chronic sialadenitis; usually in only one gland.

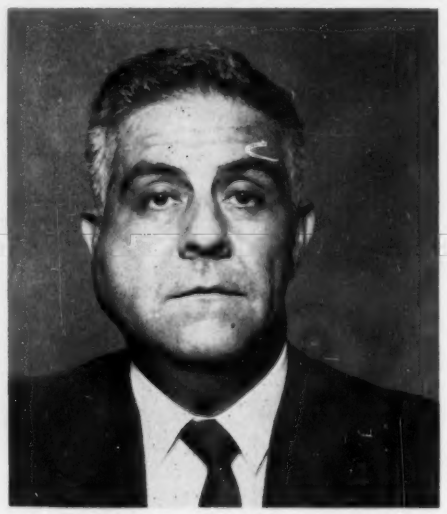


Fig. 14.—Subacute sialadenitis with abscess formation secondary to actinomycosis.

Fig. 15.—Mikulicz's disease. Diffuse enlargement of parotid gland with exaggerated lobulation on cut surface. The grayish-white color of the enlarged lobules reflects the marked lymphoid infiltrate.

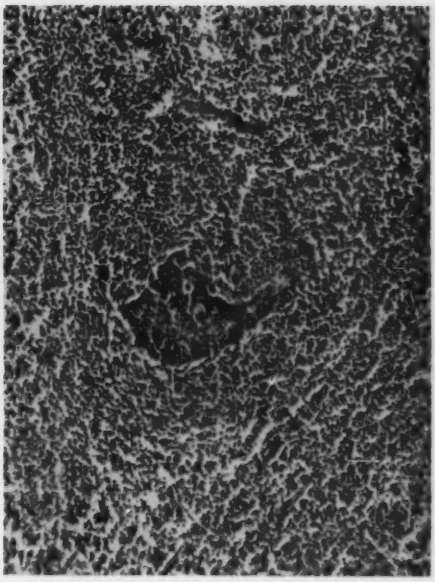


Fig. 16.—Mikulicz's disease. The parenchyma of the gland is replaced by lymphoid elements in which are seen epithelial islands that represent proliferation of ductal epithelium.

Fig. 17.—Boeck's sarcoid. The typical histologic reaction is formation of noncaseous tubercles containing giant cells.

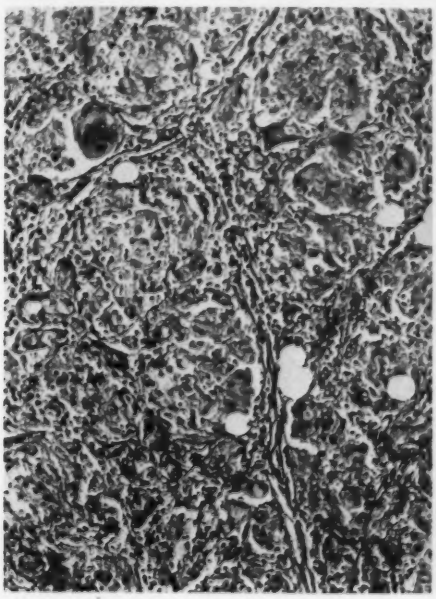
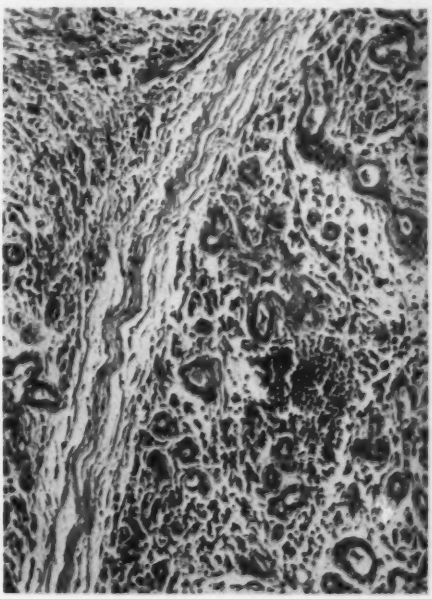


Fig. 18.—Chronic sialadenitis. This is a nonspecific inflammatory reaction with associated loss of parenchyma and compensatory fibrosis.



Moderately Malignant Tumors of the Parotid Gland

1. Cylindroma (adenoid cystic carcinoma) is a slowly growing, infrequent carcinoma (16% of the malignant series); it infiltrates nearby structures, often causing local pain or facial paralysis. 2. Acinic cell carcinoma (15%) is a slowly growing, malignant neoplasm, soft, rounded or encapsulated, presenting as a single nodule; recurrent lesions are frequent and usually multiple. 3. Low-grade mucoepidermoid carcinoma (27%) is a slowly growing neoplasm; clinically, it may initially resemble mixed tumor, with painless swelling; recurrent tumors are frequent, but distant metastasis is rare.

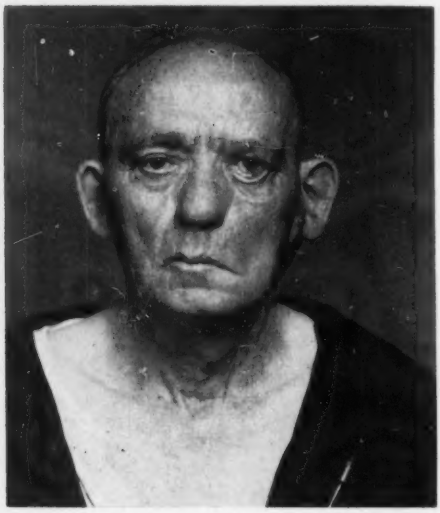


Fig. 19.—Cylindroma of left parotid gland with involvement of facial nerve.

Fig. 20.—Mucoepidermoid carcinoma that recurred three months after local excision.

Fig. 21.—Adenoid cystic carcinoma (cylindroma). The parotid gland is involved extensively. The ill-defined margins emphasize the infiltrative quality of the tumor.

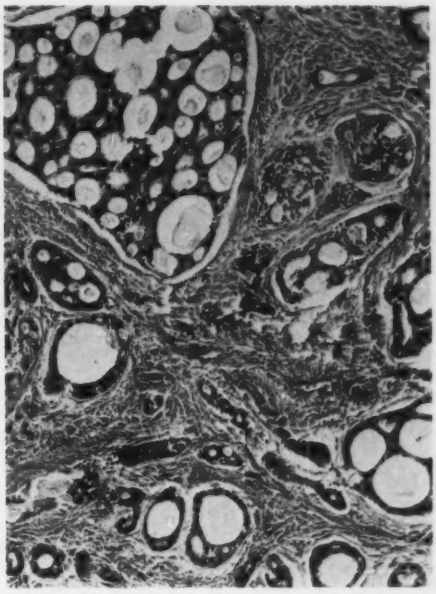


Fig. 22.—Adenoid cystic carcinoma. Small, rather regular, carcinoma cells are arranged in cords, strands or a cystic glandular (Swiss-cheese) pattern. Perineural infiltration is commonly seen.

Fig. 23.—Acinic cell carcinoma. The tumor is composed of large cells with small nuclei and slightly granular cytoplasm. They greatly resemble the acinic cells of the parotid gland.

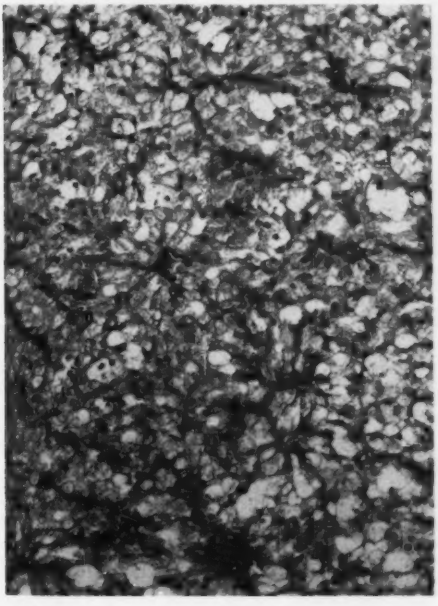
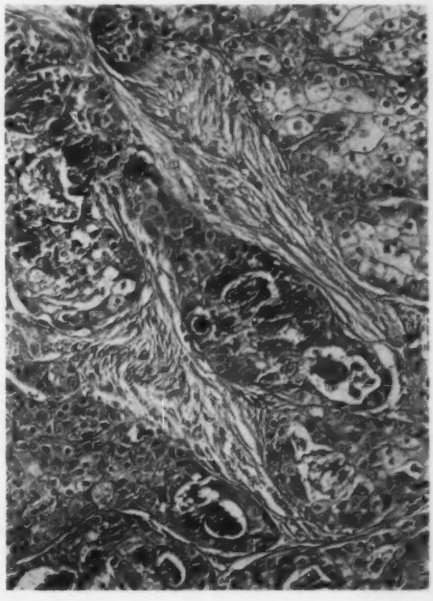


Fig. 24.—Low-grade mucoepidermoid carcinoma. Well-differentiated, mucus-producing epithelial cells are combined with squamous cells showing intercellular bridges (mucicarmine stain).



Highly Malignant Tumors of the Parotid Gland

Highly malignant carcinomas of the parotid make up about 35% of the malignant lesions. These tumors are rapidly growing neoplasms, frequently associated with pain and facial paralysis. Slightly more than one-third of the group occur as "malignant mixed tumors," representing a malignant transformation of a long-standing, benign, mixed tumor. Other histologic types include high-grade mucoepidermoid carcinoma, squamous-cell carcinoma, adenocarcinoma, and undifferentiated carcinoma. These types differ but little in their aggressive clinical behavior.

Fig. 25.—Mixed tumor of 30 years' duration with rapid enlargement and malignant transformation.

Fig. 26.—Ulcerating and rapidly growing adenocarcinoma.

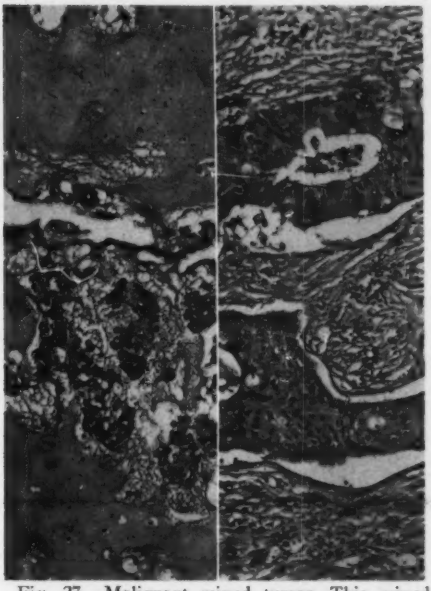


Fig. 27.—Malignant mixed tumor. This mixed tumor is composed of cartilage and epithelial elements (left), with areas of carcinoma (right) occurring in a portion of the tumor.

Fig. 28.—High-grade mucoepidermoid carcinoma. This carcinoma shows some mucous and squamous differentiation, but it is more anaplastic in appearance than the low-grade mucoepidermoid carcinoma (see Fig. 24).

Fig. 29.—Adenocarcinoma. A high-grade carcinoma is forming occasional glandular spaces. Primary squamous carcinoma is histologically similar to the metastatic lesion in the parotid shown in next section.

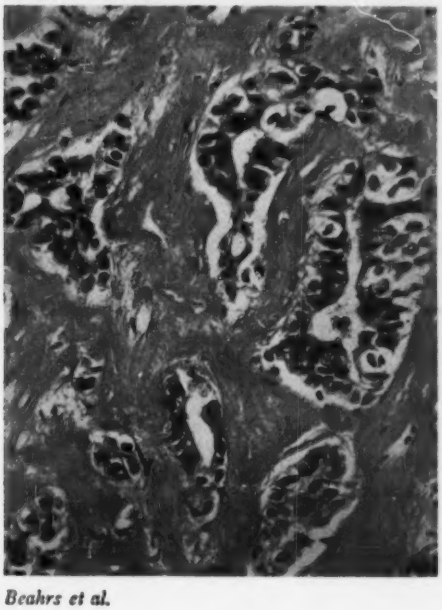
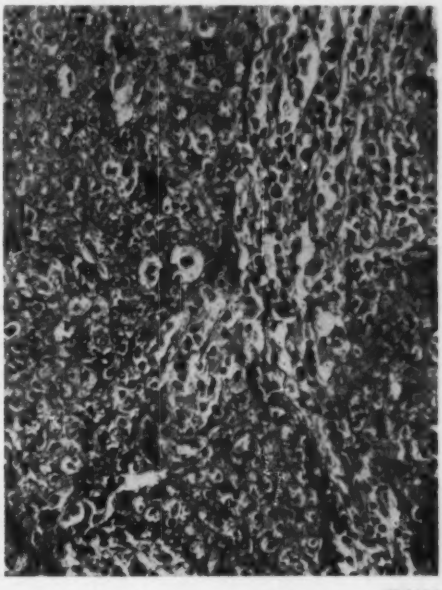


Fig. 30.—Undifferentiated carcinoma. This tumor is comprised of clumps of carcinoma cells showing no evidence of differentiation.



Metastatic and Miscellaneous Tumors of the Parotid Gland

The metastatic and miscellaneous groups of parotid tumors constitute 7% of the malignant series. 1. Metastatic carcinoma of the squamous-cell or adenocarcinomatous type may occur within the parotid or may involve the gland by direct extension from adjacent lymph nodes. 2. Malignant melanoma occurs rarely as an apparently primary phenomenon, but most frequently it represents metastatic extension from a lesion in the scalp or the face. 3. The first manifestation of lymphosarcoma may present itself as a primary parotid tumor that later may become disseminated.

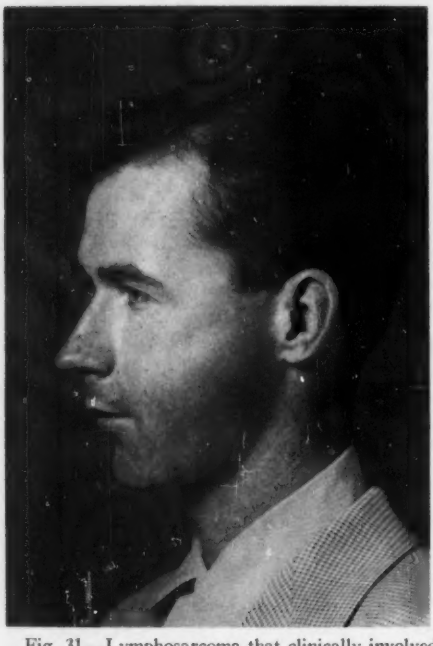


Fig. 31.—Lymphosarcoma that clinically involved only the parotid gland.

Fig. 32.—Melanoepithelioma of skin with metastasis to parotid nodes.

Fig. 33.—Metastatic malignant melanoma. This lesion in the substance of the parotid gland is extensively pigmented.

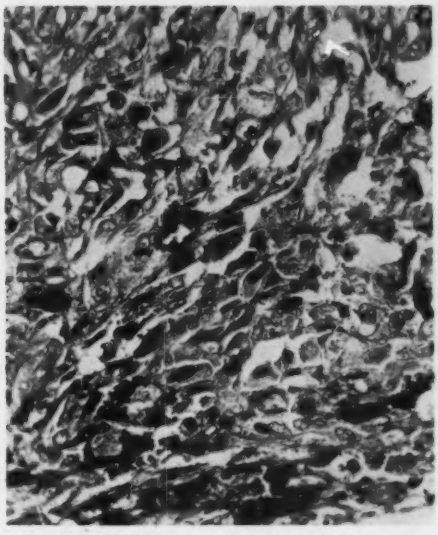


Fig. 34.—Malignant melanoma. This tumor is composed of highly anaplastic melanoma cells containing abundant melanin.

Fig. 35.—Metastatic squamous-cell carcinoma. This well-differentiated squamous-cell neoplasm involves the substance of the parotid gland.

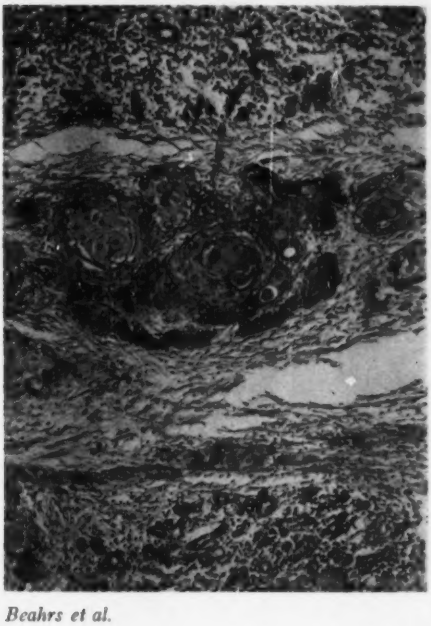
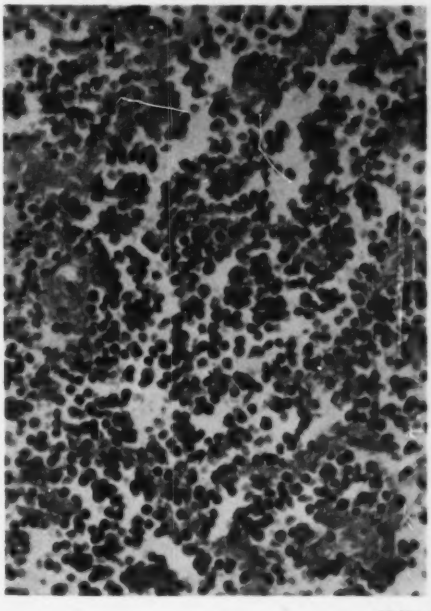


Fig. 36.—Lymphosarcoma. This is a malignant lymphoma, lymphoblastic type, occurring within the substance of the gland. Evidence of infiltration of adjacent tissues is helpful in diagnosis.



Surgical Management

Tumors of the parotid gland always should be removed completely by parotidectomy. The possibility of injury to the facial nerve has been a deterrent to such treatment in the past. Fear on the part of the surgeon of injury to this nerve has resulted in no treatment or inadequate treatment, with a resultant high rate of recurrence for both benign mixed tumors and malignant tumors, and a poor survival rate for patients with malignant lesions.

Approach to Facial Nerve

During parotidectomy, the facial nerve is best approached and identified at its main trunk, which is located medial and deep to the mastoid process in the substance of the gland. This approach has proved more satisfactory than has preliminary identification of peripheral branches, the parotid duct or the posterior facial vein.

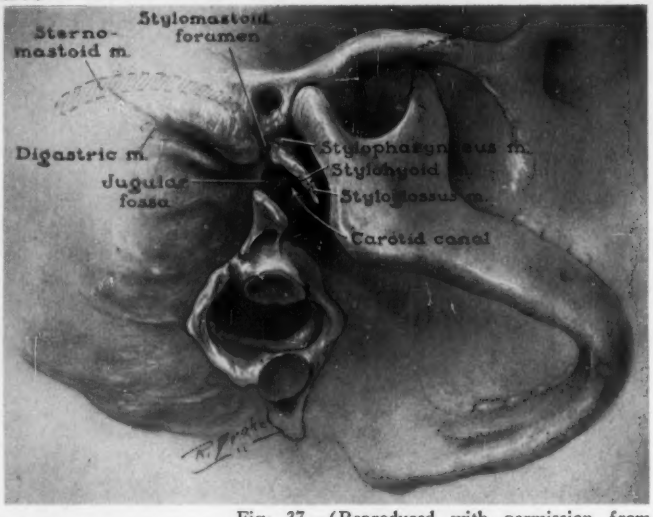


Fig. 37.—(Reproduced with permission from Beahrs, O. H., and Adson, M. A.: The Surgical Anatomy and Technic of Parotidectomy, *Am. J. Surg.* 95:885-896 [June] 1958.)

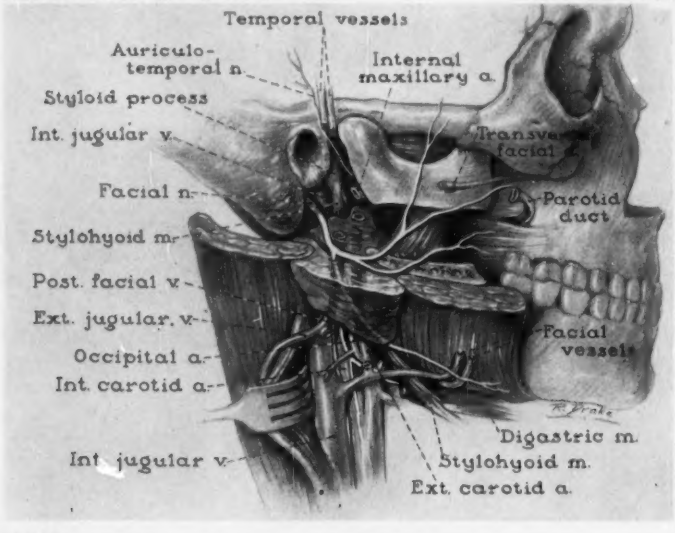


Fig. 38.—(Reproduced with permission from Beahrs, O. H., and Adson, M. A.: The Surgical Anatomy and Technic of Parotidectomy, *Am. J. Surg.* 95:885-896 [June] 1958.)

Origin of Facial Nerve

The facial nerve is consistent in its location as it emerges from the skull through the styloid foramen. As it passes through the parotid gland, it separates into two divisions and five major branches (temporal, zygomatic, buccal, marginal, and cervical). The parotid gland is not a lobar structure but a lobular organ through which the nerve branches interlace.

Subtotal Parotidectomy

For benign tumors of the parotid gland situated superficial to the facial nerve, subtotal parotidectomy is performed by removing that portion of the gland superficial to the nerve. A "Y" or inverted "T" incision is used.

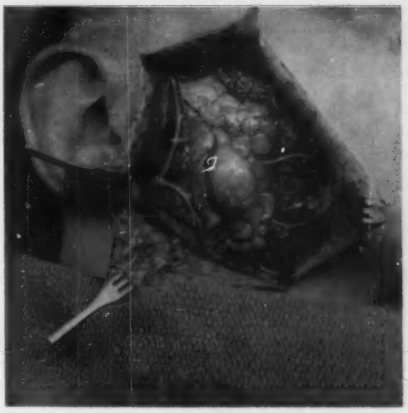


Figure 39

Total Conservative Parotidectomy

For benign tumors deep to the level of the nerve, total conservative parotidectomy is performed, with preservation of the nerve. This procedure is used also for the treatment of selected lesions of a moderate degree of malignancy.

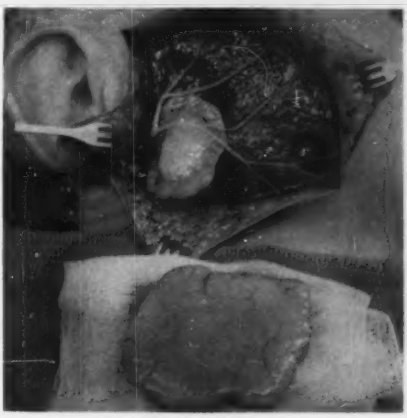


Figure 40

Total Radical Parotidectomy

For malignant lesions, total radical parotidectomy is performed, which involves sacrifice of the facial nerve. A nerve graft as shown may be inserted whenever possible to restore some degree of facial mobility.

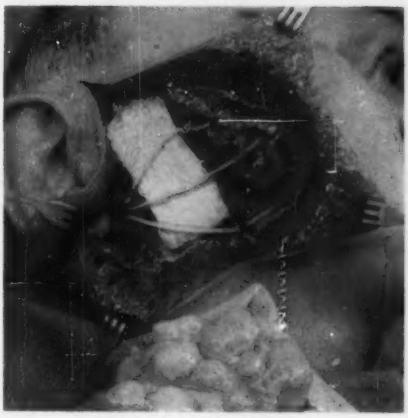


Figure 41

Results of Surgical Treatment of Malignant Parotid Tumors, 1945-1954

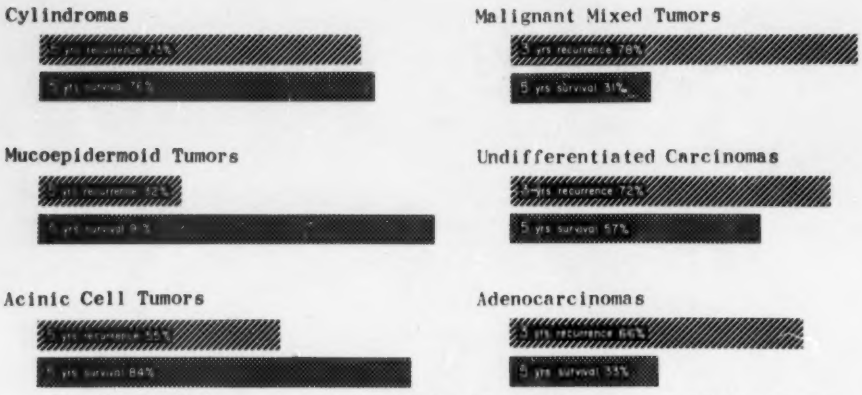
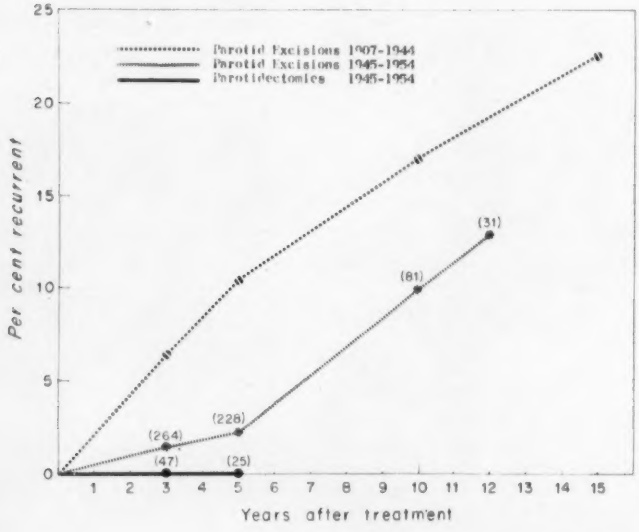


Fig. 42.—The high recurrence rate in all categories of malignant tumors and the poor survival rate in the highly malignant group are indications of the aggressive nature of these tumors.

Recurrence Rate of Mixed Tumors

Fig. 43.—The recurrence rate of mixed tumors treated by excision or enucleation has been high in the past. However, since 1949, parotidectomy has been the method of choice for treating mixed tumors, and we have had no recurrence in patients so treated.



Intracolonic Transillumination*

An Aid to the Surgeon in Localizing Polypoid Tumors of the Colon at Laparotomy

THOMAS J. FOLEY, M.D.; ALBERT G. SCHUTTE, M.D., Milwaukee, and JACK D. SELZER, M.D., Cincinnati

A patient usually submits to transabdominal polypectomy because of a roentgenologic diagnosis of a polypoid lesion of the colon which is above the reach of the sigmoidoscope or, for some reason, cannot be satisfactorily handled through the instrument. Not infrequently the surgeon is frustrated at laparotomy by his inability to localize the lesion by palpation of the colon. The smaller and/or softer polypoid tumors are more difficult to feel. Lesions less than 1 cm. in diameter seldom can be felt, while lesions 1 cm. or more in diameter usually can be felt provided they have a relatively firm consistency. Lesions up to 2 cm. or more in diameter may be missed if their consistency is relatively soft.

Colotomy with colonoscopy is a direct method of visualizing polypoid lesions regardless of their size or consistency. However, the use of this procedure is not as yet a common practice in the general hospitals. Because colotomy with colonoscopy is not employed frequently enough as a part of the surgeon's technique in cases scheduled for colon polypectomy, it was decided to evaluate the use of intracolonic transillumination at laparotomy in the localization of polypoid lesions of the colon.

A transilluminating instrument was devised (Fig. 1). The instrument is inserted through the anal canal and held at finger length in the rectum (Fig. 2) by an assistant, so that the surgeon can grasp it when ready to palpate and transilluminate the colon. After grasping the lighted instrument he has complete control of it and can manipulate it freely within the colon.

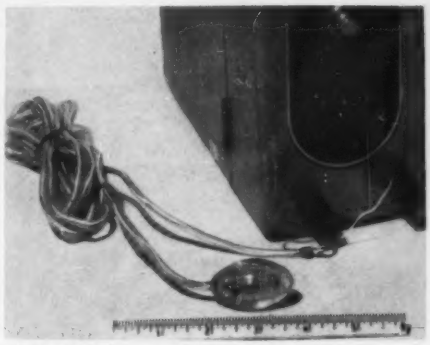


Fig. 1.—The transilluminating instrument is a water-tight, olive-shaped 2×3.5 cm. plexiglass casing containing an ordinary 6 volt flashlight bulb. The plastic cord is covered with polyethylene tubing. A battery box or spark-proof rheostat serves as the source of current. The bulb and cord may be immersed in sterilizing solution.

Findings of Limited Study

1. A thoroughly clean colon is essential to the effective use of the transilluminating instrument.

2. Polypoid lesions as small as 0.5 cm. have been transilluminated and/or palpated with the instrument.

3. Lesions less than 0.5 cm. in diameter often cannot be transilluminated or palpated against the instrument. This is particularly true when the colon is unusually fat or when the lesions are located in the mesenteric aspect.

4. The presence of the instrument within the lumen facilitates palpation of the larger soft polypoid lesions, and these bulky tumors usually can be localized by transillumination.

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From Marquette University Medical School and Milwaukee County General Hospital.

* Surgery illustrated. Shown as a scientific exhibit of the Section on Gastroenterology and Proctology at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

5. An abnormally rigid and narrow rectosigmoid, adhesions of the loops of the pelvic colon, or an unusually high splenic flexure can prevent the passage of the instrument through these areas, thereby hindering or limiting the examination.

6. Limited experience shows that the use of this intracolonic transilluminating device can be a safe aid to the surgeon in locating polypoid tumors at transabdominal polypectomy. However, this is an indirect method and is not as thorough an aid to the surgeon as the use of the direct method of colotomy with colonoscopy.

331 W. Wisconsin Ave. (8).

Fig. 3.—Shadow cast by 6×11 mm. polyp of upper pelvic colon during intracolonic transillumination.

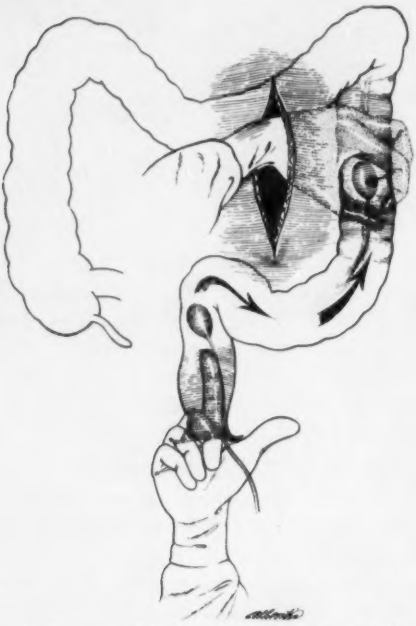

Fig. 2.—The abdomen has been opened and explored; the surgeon has taken the instrument from the rectum and is shown in complete control of it. He carefully transilluminates and palpates the colon as he milks the instrument through the organ with the fingers of one or both hands. At completion of the examination the instrument is easily withdrawn by pulling on the cord under the drapes.

Fig. 4.—Polyp transilluminated in Figure 3 is shown delivered through colotomy prior to its excision.

Fig. 5.—Excised lesion shown in Figure 4 measures about 6×11 mm. with a pedicle about 15 mm. long.

Malignant Carcinoid Syndrome

HARWELL WILSON, M.D., and EDWARD H. STORER, M.D., Memphis

The malignant carcinoid syndrome is manifested by (1) paroxysmal flushing, (2) chronic diarrhea, (3) respiratory distress, and (4) valvular heart disease. These abnormalities result from a marked increase in the amount of circulating serotonin.

The abnormal clinical findings in this syndrome result principally from the action of serotonin on the smooth muscle of the respiratory tract, the gastrointestinal tract, and the arterioles. The pathogenesis of the cardiac lesions remains obscure.

Serotonin, or 5-hydroxytryptamine, is produced by the argentaffin cells of the gastrointestinal tract as a normal metabolic product of tryptophan metabolism. Tumors of the argentaffin cells—"carcinoids"—may produce tremendous amounts of serotonin, but since the liver metabolizes serotonin to "inactive" 5-hydroxyindoleacetic acid, symptoms of hyperserotonemia result only when serotonin in large amounts is present in the systemic circulation. Symptoms of the malignant carcinoma syndrome follow hepatic metastases, metastases elsewhere drained by the systemic veins, or from primary carcinoids outside the portal area.

Malignant carcinoid should be considered in the differential diagnosis of any patient with (1) cutaneous vasomotor phenomena, (2) valvular heart disease involving the right side, (3) hepatomegaly, and (4) low-grade malignancy of the gastrointestinal tract. The diagnosis is confirmed by finding abnormal amounts of 5-hydroxyindoleacetic acid in the urine.

Remarkable palliation may be obtained by surgical removal of the serotonin-producing carcinoid metastases. This is illustrated by the following case:

A 41-year-old white man (Patient 1) was first seen in July, 1956, complaining of weight loss and diarrhea for the past 10 months. Exploratory celiotomy revealed "inoperable adenocarcinoma with liver metastases." His benign subsequent course and onset of peculiar flushing attacks aroused suspicion. The urinary 5-hydroxyindoleacetic acid was tremendously elevated (Fig. 1). At reoperation in February, 1957, multiple carcinoids of the ileum and 1,182 gm. of involved liver were resected. When last seen, 27 months after the palliative liver resection, he continued asymptomatic and was working full time as a dairy farmer

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From the Department of Surgery, University of Tennessee, College of Medicine.

* Surgery Illustrated. Shown as a scientific exhibit of the Section on Gastroenterology and Proctology at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

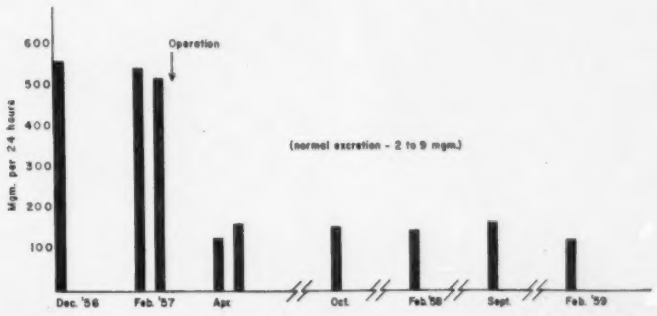

Fig. 1.—Patient 1. Quantitative 24-hour urinary excretion of 5-hydroxyindoleacetic acid.

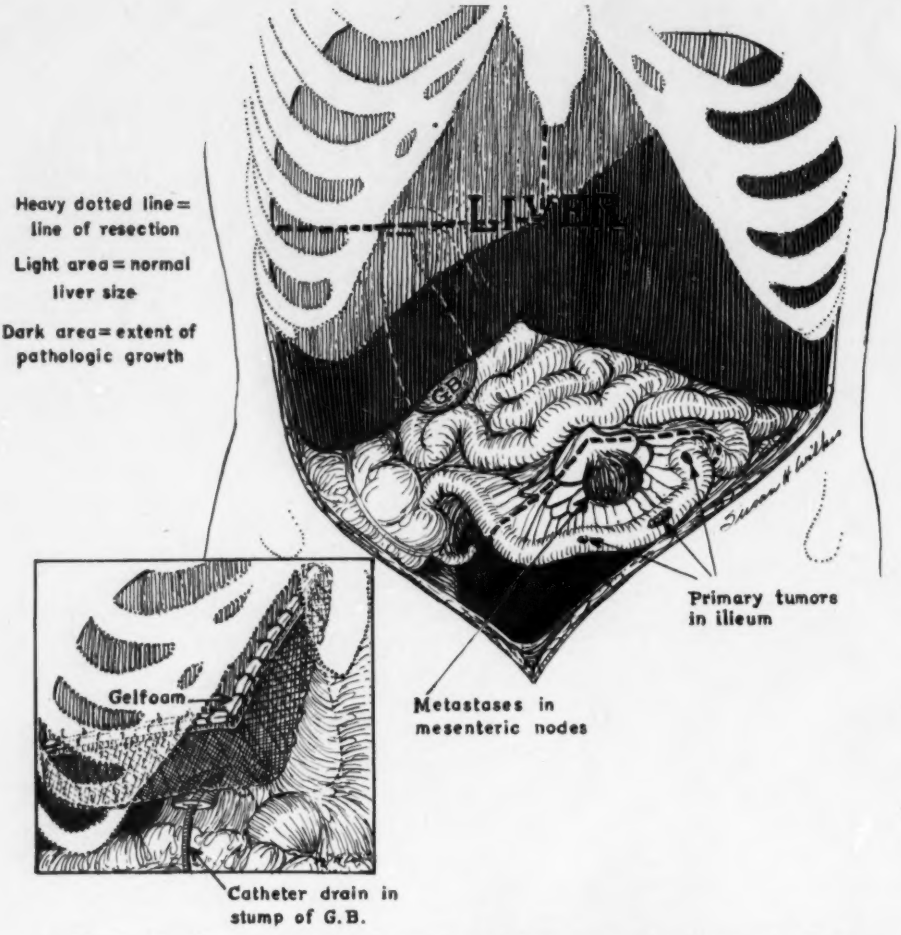
Heavy dotted line = line of resection
Light area = normal liver size
Dark area = extent of pathologic growth
LIVER
GB
Primary tumors in ileum
Metastases in mesenteric nodes
Gelfoam
Catheter drain in stump of G.B.

Fig. 8.—Drawing reveals marked enlargement of liver as a result of metastatic disease. Resection of primary tumor with mesenteric nodes and massive resection of liver removing 1,182 grams of hepatic tissue done at one sitting. Insert reveals appearance of liver after massive hepatic resection.

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1. Wilson, H., and Butterick, O. D., Jr.: Massive Liver Resection for Control of Severe Vasomotor Reactions Secondary to Malignant Carcinoid, Ann. Surg. 149:641 (May) 1959.

2. Wilson, H.: Malignant Carcinoid Syndrome: Massive Liver Resection for Symptomatic Relief, Am. Surgeon 25:567 (Aug.) 1959.

Division of Surgery, University of Tennessee.

Fig. 2.—Patient 1, exhibiting the characteristic violaceous flushing.

Fig. 3.—Patient 1. Surgical specimen showing multiple primary carcinoids of the ileum and large mass of mesenteric lymph nodes containing metastases.

Fig. 4.—Patient 1. Surgical specimen of resected liver with massive carcinoid metastases.

Fig. 5.—Patient 2. Autopsy specimen showing primary carcinoid in bronchus. The malignant carcinoid syndrome may result from tumors in this location.

Fig. 6.—Patient 3. Autopsy specimen with right ventricle open, showing a thickened, somewhat stenotic, tricuspid valve.

Fig. 7.—Patient 3. Autopsy specimen demonstrating the thickened cusps of the pulmonic valve.

2

3

4

5

6

7

Primary Repair of Defects Following the Surgical Removal of Tumors of the Face*

JOEL J. PRESSMAN, M.D.; WALTER BERMAN, M.D., and MILDRED BURTZ SIMON, R.N., Los Angeles

Introduction

This exhibit, which was originally presented in backlighted color transparencies, is concerned with the repair of surgical defects resulting from wide excision of tumor-bearing areas in and about the face. These can be closed by a variety of methods, but whenever possible the use of adjacent tissue of full thickness in the form of a sliding flap, which carries its own blood supply, is far and away the most desirable. This not only assures proper matching of color, texture, and thickness, but normal reactions to cold, heat, and sun corresponding to those of the surrounding area. Healing is by primary union and takes place rapidly.

Examples of such use of adjacent tissue for primary repair about the face, and details of surgical techniques involved are demonstrated in a series of cases. As part of the exhibit, viewers were given a descriptive pamphlet including the description of data presented herein.

The first three cases represent the repair of defects by the use of sliding flaps, each of which presents separate and distinct problems as follows:

1. Repair by sliding flaps when one wound margin is too long (Case 1)
2. Repair by sliding flaps when one wound margin is too short (Case 2)
3. Repair by sliding flaps when the excess of skin is not adjacent to the wound (Case 3)

Advantage can be taken of the principle of the sliding flap to reposition structures such as a residual segment of eyebrow, and this is illustrated in Case 4, which also demonstrates the use of a double sliding flap, created when it is desirable to use skin on both sides of the surgical defect.

One example of permissible use of a full-thickness skin graft is presented (Case 5).

Four surgical procedures (each having many modifications) are useful in the surgical treatment of malignant and premalignant lesions of the lips. These consist of mucosal stripping for premalignant hyperkeratosis (Case 6), the V-excision for smaller malignancies (Case 7), a combination of the above two when cancer has developed in one area of a more diffuse premalignant process (Case 8), and a fourth operation for extensive lesions in which a portion of the uninvolved lip is swung as a flap for closure of defects too wide to permit direct suturing of the residual segments of the involved lip (Case 9).

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From the City of Hope National Medical Center and the Department of Surgery, University of California at Los Angeles School of Medicine.

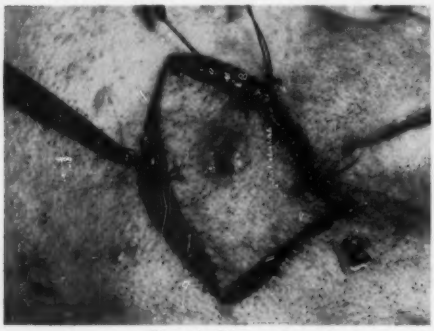
*Surgery Illustrated. Shown as a scientific exhibit of the Section on Laryngology, Otology, and Rhinology at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959. A demonstration of resection and repair of various lesions of the external ear was also included in the original exhibit as well as a technique for repair of unsightly, depressed tracheotomy scars. These are being published in the *A.M.A. Archives of Otolaryngology.*

Case 1

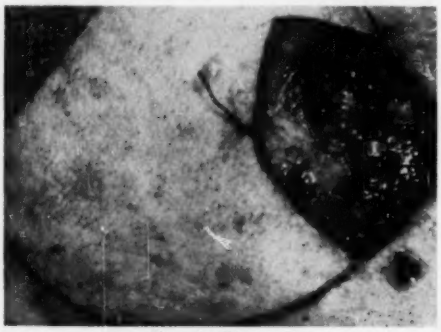
Triangular Excision.—Figure 1 illustrates the use of a triangular excision with extension of the base in a gentle rocker curve to create a sliding flap.

Traction on the mouth and nose is avoided by creating the flap from the redundant tissues of the cheek and neck posteriorly. Scarring is minimized by placing the rocker segment of the incision along which the flap will slide in the direction of Langer's lines (Fig. 2). The flap is then elevated, and the skin of the area undermined (Fig. 3).

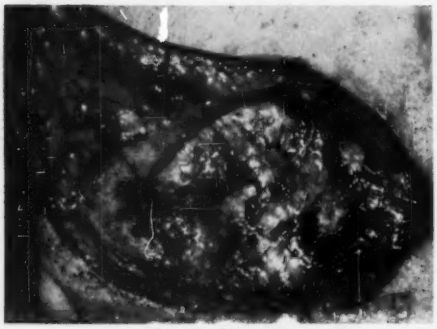
Unequal wound margins are sutured by accurately approximating the skin edges beginning anteriorly, thereby throwing all the redundancy of the longer element posteriorly, and thus creating a "dog-ear," which is seen in the lower left corner (Fig. 4). This is then converted into a triangular segment by a small curved incision (Fig. 5). The excess is delineated with a marking of dye, the position of which is established by tucking the flap under (Fig. 6). The excess is then removed. The edges then approximate exactly. Suturing is in layers (Fig. 7). The result after two weeks is illustrated in Figure 8.



Case 1, Figure 1



Case 1, Figure 2



Case 1, Figure 3

Case 1, Figure 4

Case 1, Figure 5

Case 1, Figure 6

Case 1, Figure 7

Case 1, Figure 8

Case 2

Repair by Sliding Flaps When One Wound Margin Is Too Short.—Recurrence of carcinoma is demonstrated in a previously irradiated widespread area of the cheek (Fig. 1). The entire region, extending from a level above the eyebrow to below the lips is excised en masse (Fig. 2). Repair is by a sliding flap prepared from the skin posteriorly. The length of the medial margin of the wound is, however, greater than the lateral element, so satisfactory approximation of the wound edges cannot be accomplished except by lengthening the lateral wound margin. The upper limit of the flap (Fig. 3) is delineated by a transverse incision at *A*. The lower limit, where the skin is more redundant, is prepared by using a combination of vertical and transverse incisions, which is demonstrated inferiorly. This appreciably increases the length of the lateral skin margin, which now corresponds to that of the medial margin. By undermining, the flap can be mobilized and transposed so that point *B* approximates point *C* and the remainder of the wound edges fall into a matching position (Fig. 4). The deeper tissues are sutured, fixing the flap in position (Fig. 5), and the skin is closed (Fig. 6). Good healing takes place after 10 days (Fig. 7). The final cosmetic result after one year is demonstrated in Figure 8. A radical neck dissection was eventually required, but no local recurrence was in evidence.

Case 2, Figure 1

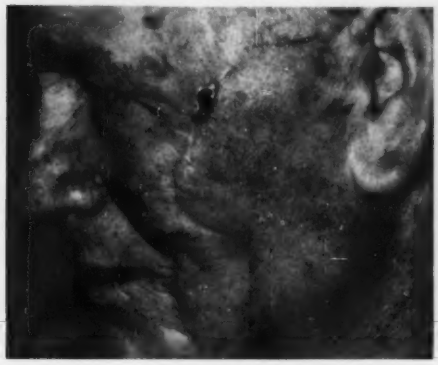
Case 2, Figure 2

Case 2, Figure 3

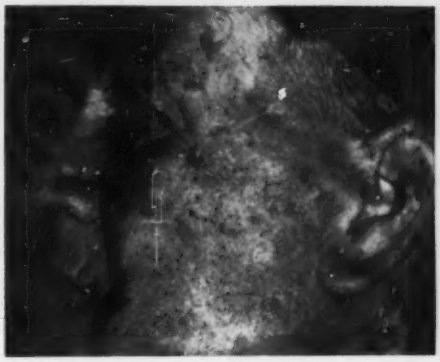
Case 2, Figure 4

Case 2, Figure 5

Case 2, Figure 6



Case 2, Figure 7



Case 2, Figure 8

Case 3

Sliding Flaps Where Available Skin Is Not Adjacent to the Excised Area.—The principle demonstrated is that of creating and utilizing sliding flaps where no excess or deficiencies of skin exist, but where the available skin is not adjacent to the excised area.

A primary local flap is elevated and slid into position, unavoidably creating a defect at the donor site. This is in turn closed by a second flap taken from where excess skin is more readily available. The illustration is a case of two coexisting lesions involving the left ala of the nose (Fig. 1) and the preauricular area including the contiguous portion of the auricle (Fig. 2). Both lesions are widely excised (Figs. 3 and 4). The primary flap carrying its own blood supply (the infraorbital artery) is partially created by an inferiorly placed curved incision (*A*) which joins the two wounds (Fig. 5). It is completed and mobilized by the utilization of a superiorly placed transverse incision (*B*). The flap is then undermined and moved forward to fill the defect about the nose. A new defect is thus created posteriorly (Fig. 6). It is, in turn, filled by a second sliding flap (*C*), brought up from the redundant skin of the neck below (Fig. 7). A small area of the auricle itself is covered by a split skin graft (*D*) taken from behind the ear (Fig. 8). The postoperative cosmetic result after two years is illustrated in Figure 9.

Case 3, Figure 1

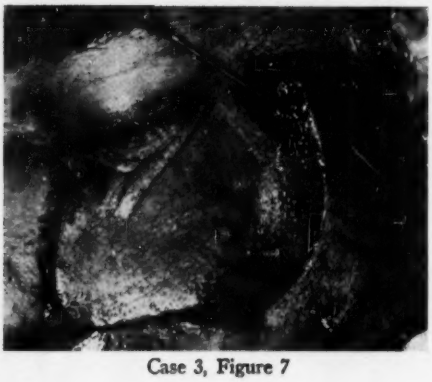
Case 3, Figure 2

Case 3, Figure 3

Case 3, Figure 4

Case 3, Figure 5

Case 3, Figure 6



Case 3, Figure 7

Case 3, Figure 8

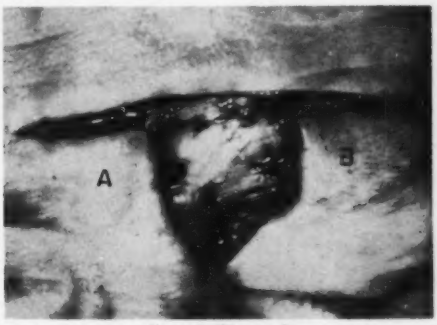
Case 3, Figure 9

Case 4

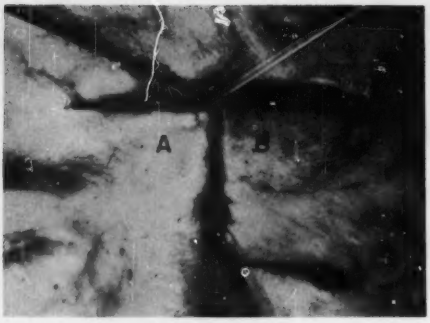
Use of a Double Flap.—This is primarily a demonstration of the use of a double flap to permit utilization of skin from both sides of a defect.

The example is that of a basal-cell carcinoma in the region of the eyebrow removed by a triangular excision (Fig. 1). The case demonstrates two points: (1) the use of a sliding flap for repositioning a residual segment of eyebrow for better cosmetic effect, and (2) the use of a double rocker extending from each side of the base of a triangular excision to provide two flaps, *A* and *B*, to permit the use of skin from both sides of a wound for covering the defect (Fig. 2). After the flaps are undermined, they are brought together (Fig. 3). The "dog-ear" of excess tissue (*C*) unavoidably created (Fig. 4) whenever skin edges of unequal length are sutured without puckering of one side is subsequently eliminated by converting it into a small triangle (Fig. 5). This excess is delineated with dye (Fig. 6) and removed. The result after two weeks is demonstrated in Figure 7 and the final cosmetic result after two years in Figure 8. Note the normal position of the eyebrow and the absence of scarring which results from positioning the incision so that closure will correspond to the direction of Langer's line.

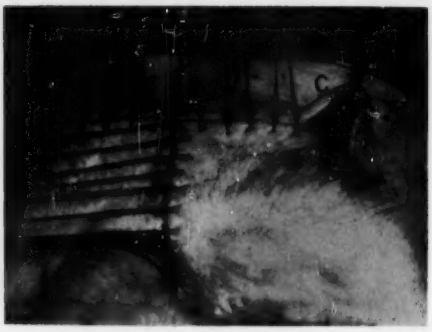
Case 4, Figure 1



Case 4, Figure 2



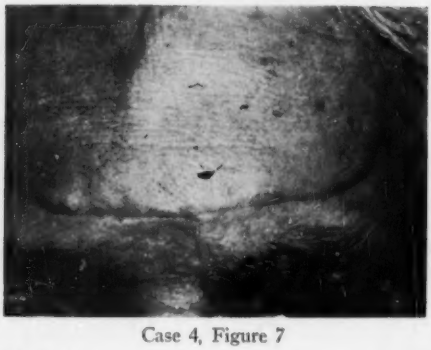
Case 4, Figure 3



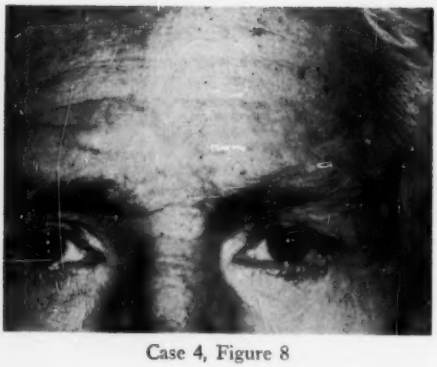
Case 4, Figure 4

Case 4, Figure 5

Case 4, Figure 6



Case 4, Figure 7



Case 4, Figure 8

Case 5

Use of Full-Thickness Grafts.—This case demonstrates one of the instances in which the use of full-thickness grafts provides an acceptable (but not perfect) repair.

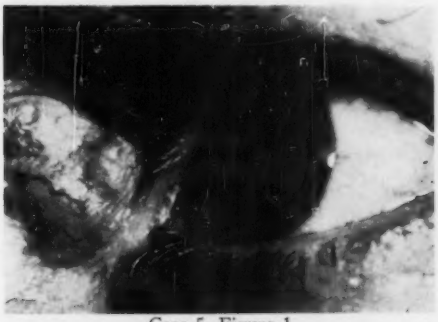
Free skin grafts, especially split-thickness grafts taken from distant sites, are ordinarily unsuitable for use upon the face except as a temporary expedient. The skin seldom matches, and a "tire patch" effect is created. In those areas where the use of a small free graft is advantageous, a full thickness of skin taken from the upper eyelid provides the best cosmetic result, since it more nearly matches the surrounding tissue than skin from distant areas. It is, however, often not thick enough to replace sliding grafts in many areas, so its use should be limited to specific sites where a depression will not result.

A surprisingly large segment of skin can be obtained in this manner. The donor site heals without a scar. The removal of excess skin from the donor eyelid improves the appearance, and the operation is often done on the opposite eyelid as well, solely for cosmetic reasons.

The example used is of a sharply circumscribed lesion of low-grade malignancy arising from the skin of the lateral nasal wall near the inner canthus of the eye. At the time of surgery, all margins of the wound examined by frozen section were negative for cancer cells.

If the defect had been larger, a tube flap from the forehead would have been used.

Figure 1 is the lesion near the inner canthus of the left eye; Figure 2, removal of the lesion; Figure 3, outline of incision at donor site; Figures 4 and 5, removal of full thickness of skin of upper eyelid; Figure 6, donor site sutured; Figure 7, skin graft sutured in situ. Long sutures are to retain pressure dressings. Note perforation for drainage; Figure 8, pressure dressings in situ; Figure 9, cosmetic effect at donor site; Figure 10, healing at area of excision after six months.



Case 5, Figure 1

Case 5, Figure 3

Case 5, Figure 2

Case 5, Figure 4

Case 5, Figure 5

Case 5, Figure 7

Case 5, Figure 8

Case 5, Figure 6

Case 5, Figure 9

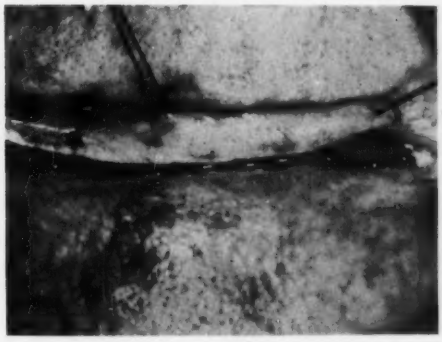
Case 5, Figure 10

Case 6

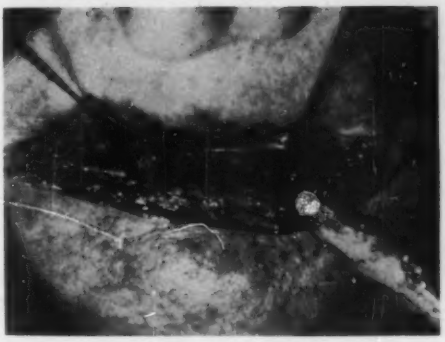
Mucosal Stripping for Premalignant Hyperkeratosis or Leukoplakia.—Premalignant hyperkeratosis and leukoplakia of the lip can be resected and the lip repaired by a sliding mucosal flap from the inner surface of the lip. It is well to omit excising the commissures if possible, since suture lines at this site are undesirable. The mucosa must be freed well down into the labiogingival gutter, and an even, precise suture line established at the carmine border. This is an effective method for preventing the development of cancer of the lip arising from premalignant lesions.

Figure 1 demonstrates the premalignant changes. Figure 2 shows the incision carried through the mucosa. Figure 3, the strip of involved mucosa is removed down to the orbicularis ori muscle. Figure 4, the buccal surface of the residual mucosa is freed to the lower limit of the mandible, creating a mucosal flap. Figure 5, the mucosal flap is elevated and sutured to the skin margin. Figure 6, the suture line is completed. Figure 7 shows the end-result after a few weeks. The cosmetic result constantly improves over a long period of time.

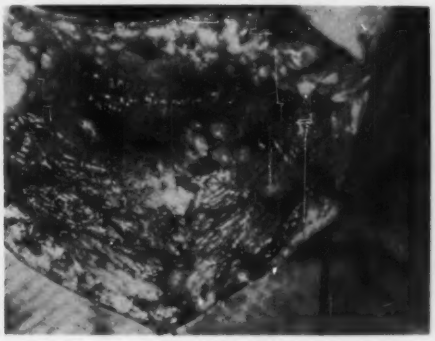
Case 6, Figure 1



Case 6, Figure 2

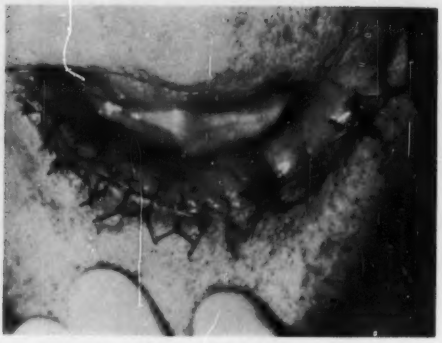


Case 6, Figure 3



Case 6, Figure 4

Case 6, Figure 5



Case 6, Figure 6

Case 6, Figure 7

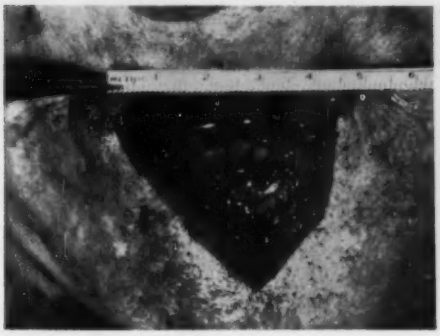
Case 7

V-Excision.—Localized squamous-cell carcinoma is resected by a V-excision and repaired by side-to-side approximation. Owing to flaccidity of the structures about the mouth, closure of a large defect (in this instance 4.5 cm.) is possible without producing functional disability or a "fish-mouth" effect. In the patient shown, the surgery is recent and the scar more noticeable than it will be later on. Figure 1 demonstrates the lesion; Figure 2, the outline of the area to be excised; Figure 3, traction sutures being used for fixation. The fingers squeezing the lip occlude the vessels to provide a dry surgical field. Figure 4 demonstrates the through-and-through V-resection; Figures 5 and 6, suturing in layers including the intraoral mucosa; Figures 7 and 8, the cosmetic result a few weeks postoperatively. Further improvement in the appearance of the scar is to be anticipated.

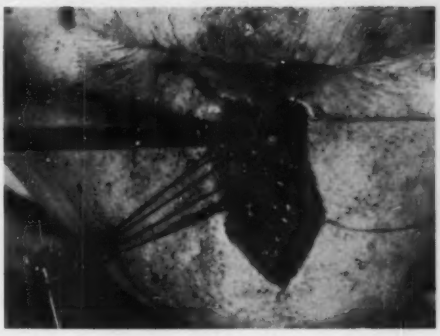
Case 7, Figure 1

Case 7, Figure 2

Case 7, Figure 3



Case 7, Figure 4



Case 7, Figure 5

Case 7, Figure 6

Case 7, Figure 7

Case 7, Figure 8

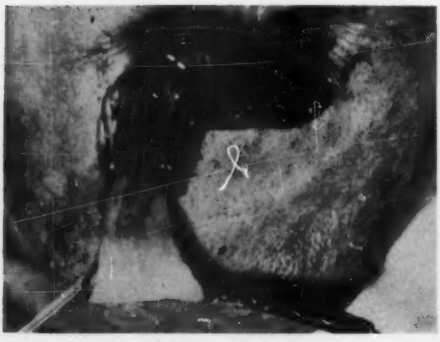
Case 8

Combined V-Excision and Stripping.—The second case demonstrates en bloc stripping for premalignant changes, and V-excision in continuity of the site where carcinoma has already developed. The stripped area should be carefully examined histologically for evidence of malignant degeneration elsewhere in the tissue. Figure 1 demonstrates the diffuse hyperkeratosis with malignant degeneration above the V. The area of excision is outlined; Figures 2 and 3 are of the through-and-through V-resection at the tumor site with the attached, stripped mucosa removed in continuity. Figure 4 shows the mucosa internal to the stripped surface being freed to the lower limit of the mandible, forming a mucosal flap. In Figure 5 the mucosa is elevated to be sutured to the skin margin. Figure 6, the internal aspect of the V-excision is sutured mucosa to mucosa; Figure 7, suturing completed in layers; Figure 8, the result two months postoperatively.

Case 8, Figure 3

Case 8, Figure 1

Case 8, Figure 4



Case 8, Figure 2

Case 8, Figure 5

Case 8, Figure 6

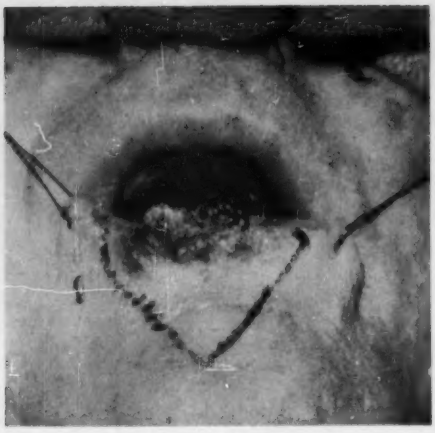
Case 8, Figure 7

Case 8, Figure 8

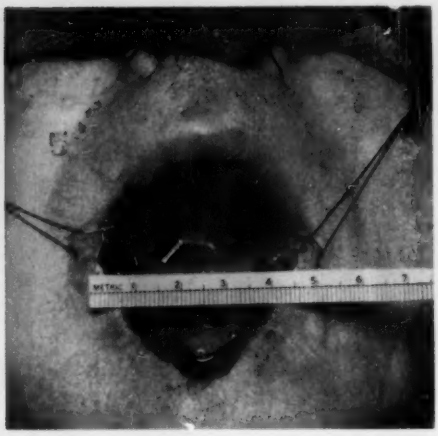
Case 9

Abbé-Estlander Flap.—The series demonstrates one of many versions of the Abbé-Estlander flap used for the surgical treatment of extensive cancer of the lip. The basic principle is the utilization of a segment taken from the uninvolved lip, and swung as a pedicle flap to close a defect too wide for primary side-to-side approximation. The donor site selected for the flap is important, and a trial pattern is used in order that, when swung down, the flap will advantageously fill the defect and not be too far to one side or the other. It survives by reason of the incorporated labial artery which anastomoses freely enough with its fellow so that the vascular supply of either side of the lip is adequate for both sides. If the vessel is incorporated in the pedicle and uninjured, the transplanted flap will survive. The pedicle remains attached for several weeks until it has taken up an independent blood supply. It is then severed.

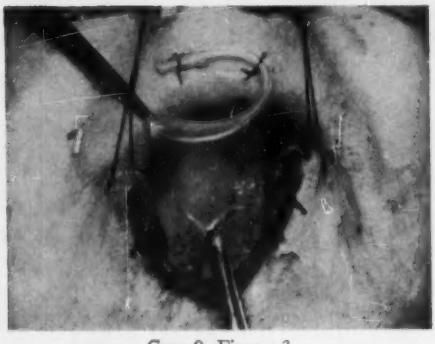
Figure 1 demonstrates the lesion and outline of the excision; Figure 2 indicates the width of the excised segment is 4.5 cm. The segment to be transposed from the upper lip into this defect will be one-half this width, or 2.25 cm. This equalizes the length of the postoperative upper and lower lips. Figure 3 shows a pattern cut from the cuff of a rubber glove. The rolled edge represents the labial artery. The triangular pattern is placed in the middle of the defect. Figure 4, the pattern is placed on the upper lip and outlined, delineating a donor site which will swing, on a pedicle containing the vessel, into the proper site. Figure 5 demonstrates the outline of the flap and the vessel-containing pedicle just above the word "metric." Figure 6, the graft is cut through and through; the pedicle remains intact and the vessel is clamped laterally. Figure 7, the flap is swung into position. Figure 8, suturing is in layers from the mucosa *out*. Figure 9, the suturing is completed, and the pedicle overlies the hemostat. Figure 10, a Barton bandage is applied for a few days.



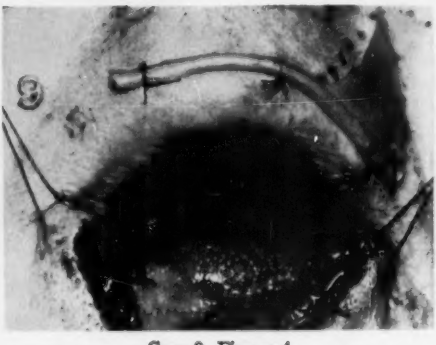
Case 9, Figure 1



Case 9, Figure 2

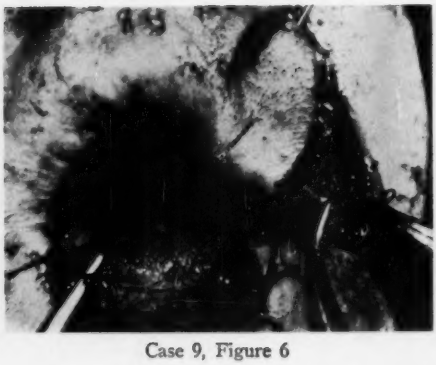


Case 9, Figure 3

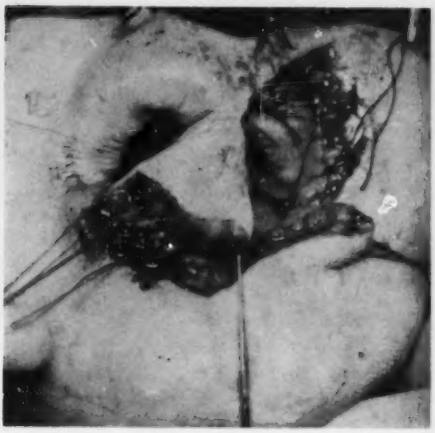


Case 9, Figure 4

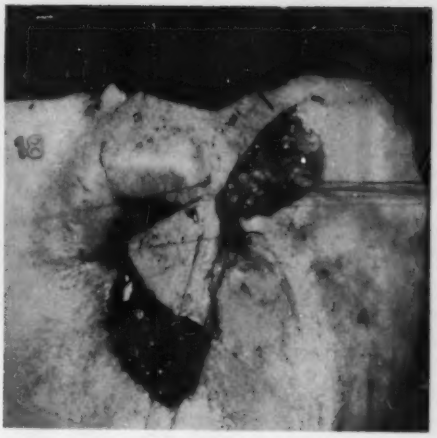
Case 9, Figure 5



Case 9, Figure 6



Case 9, Figure 7



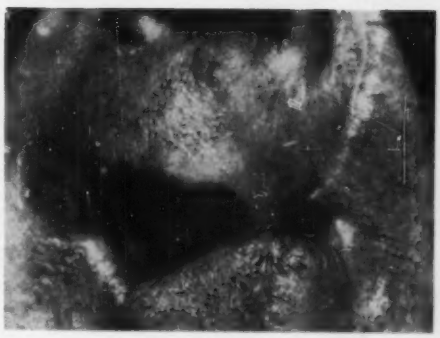
Case 9, Figure 8

Case 9, Figure 9

Case 9, Figure 10

The second stage is delayed for several weeks. Figure 11 demonstrates the pedicle still intact; Figure 12, the beginning of severing the pedicle with an underlying finger for support; Figure 13, the labial artery exposed and isolated; Figure 14, the labial artery to be clamped, tied, and cut; Figure 15, the margins of the pedicle sutured; Figures 16 and 17, end-result after one year. The size of the mouth is reduced in transverse diameter by 2.25 cm., representing one-half the width of the excised lesion.

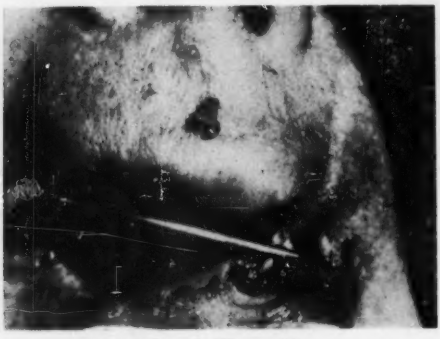
University of California at Los Angeles School of Medicine.



Case 9, Figure 11

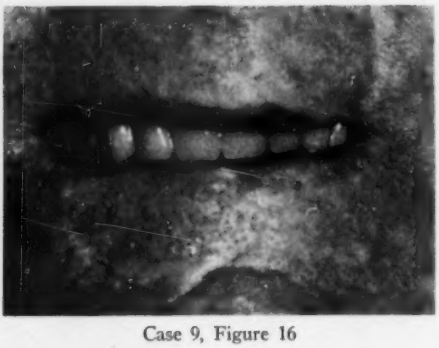
Case 9, Figure 12

Case 9, Figure 13



Case 9, Figure 14

Case 9, Figure 15



Case 9, Figure 16

Case 9, Figure 17

Calculous Disease of the Biliary Tract*

BJORN THORBJARNARSON, M.D.; FRANK GLENN, M.D., and CHARLES PEARCE, M.D., New York

Introduction

Calculous disease of the biliary tree is a common affliction. Over 10% of our adult population harbors cholelithiasis. The successful treatment of this ailment depends upon the utilization of available diagnostic aids, proper understanding of the course of the disease, and judicious application of surgical therapy.

Diagnosis by Intravenous Cholangiography

Intravenous cholangiography allows radiographic visualization of the biliary tree without the concentrating action of the gallbladder, and where oral cholecystography fails. It helps evaluate the common duct prior to cholecystectomy; this tends to lessen the incidence of retained or overlooked common duct stones. It is of utmost value in appraising symptoms following cholecystectomy such as may be caused by a re-formed gallbladder, cystic duct stump, or overlooked and new-formed common duct stones. It may also be of aid in evaluating the acute abdomen when acute cholecystitis is suspected.

525 E. 68th St., (21).

Fig. 1.—Congenital anomaly. The common duct is extremely narrow. The cystic duct is dilated and enters the duodenum separate from the common duct.

Fig. 2.—Cholecystolithiasis. Normal common duct.

Submitted for publication Aug. 24. 1959.

From The New York Hospital-Cornell University Medical Center.

* Surgery Illustrated. Shown as a scientific exhibit of the Section on Surgery, General and Abdominal, at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

Fig. 3.—Choledocholithiasis. Normal size common duct. Cystic duct obstruction.

Fig. 4.—Common duct stone. Dilated common duct.

Fig. 5.—Common duct stone. Dilated common duct.

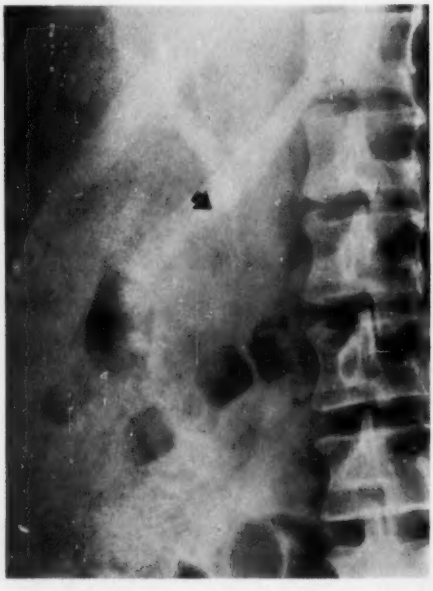


Fig. 6.—Common duct stone.

Fig. 7.—A long and narrow cystic duct remnant.

Fig. 8.—A large cystic duct remnant.

Fig. 9.—A large cystic duct remnant containing stones.

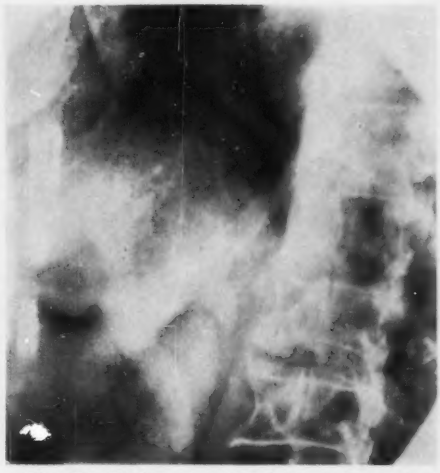


Fig. 10.—Benign stenosis of the lower end of the common duct. Dilated common duct.

Fig. 11.—Benign stenosis of the lower end of the common duct. Dilated common duct.

Fig. 12.—Calcific density in the right upper quadrant.

Fig. 13.—The calcific density is outside the biliary ductal system.

Fig. 14.—A gallstone in the right upper quadrant.

Fig. 15.—The stone is in the common duct.

Fig. 16.—A patient suspected of having acute cholecystitis. There is nonfunction of the gallbladder; the common duct is displaced toward the midline, and there is narrowing from external pressure.

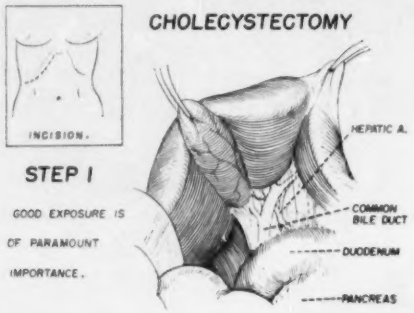
Surgery

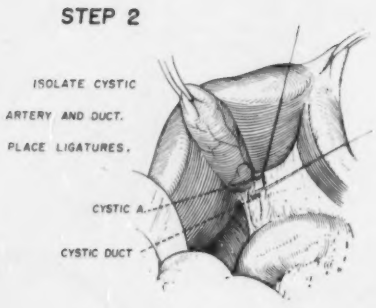
Surgery cures cholelithiasis when performed during the early stages of the disease. Operations should be performed while the stones are confined to the gallbladder and before complications arise. Prevention is better than doubtful cure.

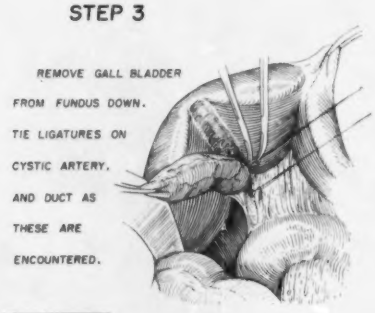
Mortality from cholecystectomy alone for chronic cholecystitis and cholelithiasis is minimal. During the past 25 years 3,227 such operations were performed; the mortality was less than 0.5%. As complications of the disease occur, mortality also increases; 801 cholecystectomies for acute cholecystitis had a mortality of 1.6%.

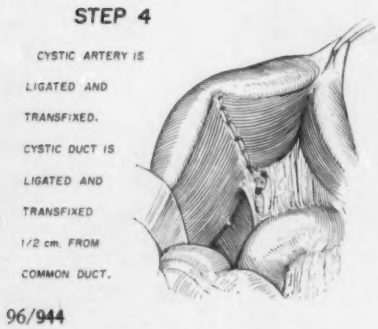
Mortality increases and symptomatic relief decreases, if surgery is delayed until the later stages of the disease. During the same period as the above group 492 cholecystectomies with choledochotomy for chronic cholecystitis, cholelithiasis and choledocholithiasis were done; mortality was 3.8%.

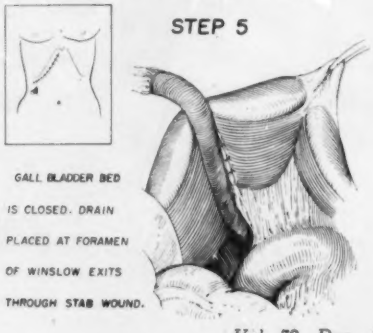
Cholecystectomy is the operation of choice for acute cholecystitis. Cholecystostomy may be life-saving, but the mortality reflects the advanced stage of the disease and the poor condition of the patient. In 114 cholecystostomies for acute cholecystitis the mortality was over 10%.











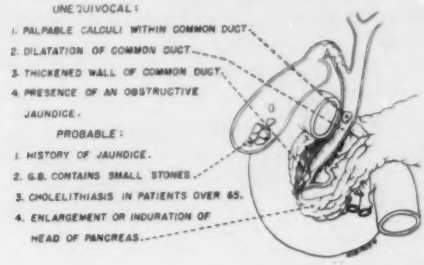
CHOLEDOCHOTOMY SHOULD BE PERFORMED UNDER THESE CIRCUMSTANCES ——

UNEQUIVOCAL:

1. PALPABLE CALCULI WITHIN COMMON DUCT.
2. DILATATION OF COMMON DUCT.
3. THICKENED WALL OF COMMON DUCT.
4. PRESENCE OF AN OBSTRUCTIVE JAUNDICE.

PROBABLE:

1. HISTORY OF JAUNDICE.
2. G.B. CONTAINS SMALL STONES.
3. CHOLELITHIASIS IN PATIENTS OVER 65.
4. ENLARGEMENT OR INDURATION OF HEAD OF PANCREAS.



CHOLEDOCHOTOMY

STEP 1

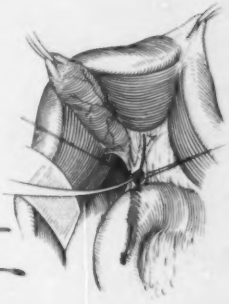
DUODENUM REFLECTED. COMMON DUCT PALPATED.

STEP 2

PROBE AND EVACUATE RIGHT AND LEFT HEPATIC DUCTS. DEMONSTRATE PATENCY OF LOWER END OF COMMON DUCT.

PROBE

SCOOP



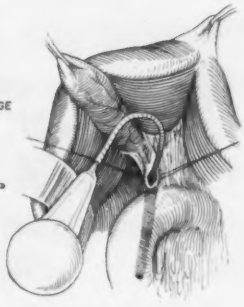
STEP 3

SYRINGE

SALINE

CATHETER

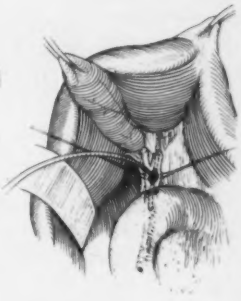
IRRIGATE THE DUCTS THOROUGHLY.



STEP 4

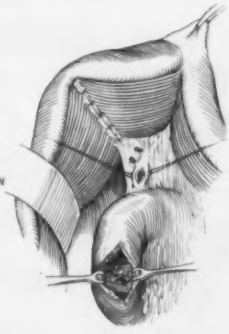
UROCON 30 %

OPERATIVE CHOLANGIOGRAM



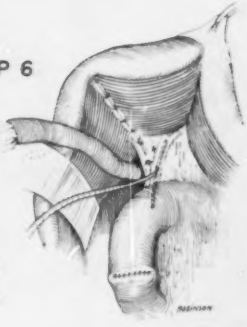
STEP 5

IF PROBING AND-OR CHOLANGIOGRAM REVEAL AN OBSTRUCTION AT THE LOWER END OF THE COMMON DUCT, DO DUODENOTOMY AND RETROGRADE EXPLORATION.



STEP 6

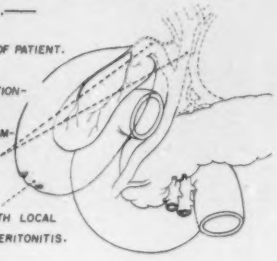
DRAIN COMMON DUCT WITH "T" TUBE. CLOSE DUODENOTOMY TRANSVERSELY. DRAIN AND "T" TUBE EMERGE THROUGH STAB WOUND.



Thorbjarnarson et al.

IN ACUTE CHOLECYSTITIS CHOLECYSTOSTOMY SHOULD BE PERFORMED UNDER THESE CIRCUMSTANCES.——

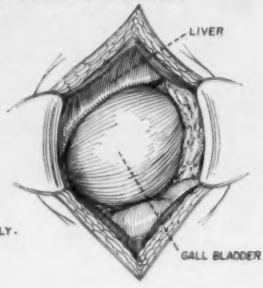
1. POOR CONDITION OF PATIENT.
2. ANATOMICAL RELATIONSHIPS OBSCURE BECAUSE OF INFLAMMATORY REACTION.
3. PERFORATION OF GALL BLADDER WITH LOCAL AND SPREADING PERITONITIS.



CHOLECYSTOSTOMY

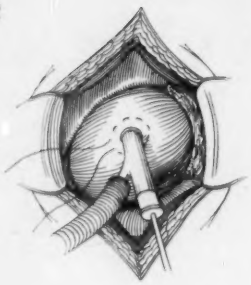
STEP 1

EXPOSE FUNDUS ONLY.



STEP 2

PLACE PURSE STRING SUTURE ASPIRATE WITH TROCAR.

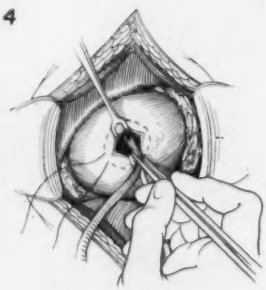


STEP 3

EVACUATE STONE WITH SCOOP.

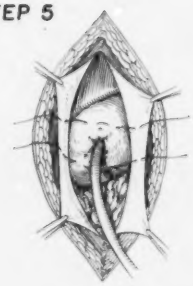
STEP 4

INSERT FOLEY CATHETER.



STEP 5

SUTURE FUNDUS TO PERITONEUM.



Classification

Calculous disease of the biliary tree is usually progressive and may be divided into stages according to its development.

Stage 1. Early Disease: Found in 10% of Surgical Patients

Fig. 35.—Gallbladder has stones, but is thin-walled and normal in outside appearance.

Fig. 36.—Common duct has no stones.

Fig 37.—Liver is grossly and histologically normal.

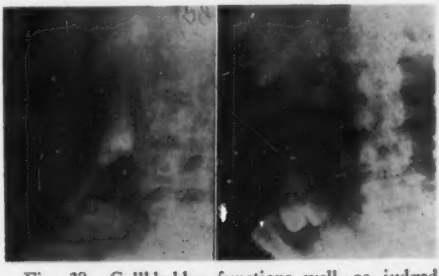


Fig. 38.—Gallbladder functions well, as judged by oral cholecystography.

Stage II. Moderately Advanced Disease: Found in 72% of Surgical Patients

Fig. 39.—Gallbladder has a thickened wall. Microscopically, there are Aschoff-Rokitansky sinuses and fibrosis of the wall.

Fig. 40.—Common duct has stones in 7% to 15%.

Fig. 41.—Liver rarely shows early gross or microscopic signs of cirrhosis.

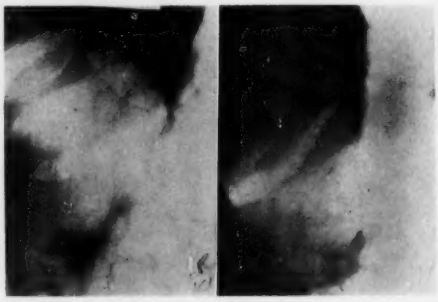


Fig. 42.—Gallbladder functions poorly on oral cholecystography.

Stage III. Advanced Disease: Found in 15% of Surgical Patients

Fig. 43.—The gallbladder is shrunken around the stones; the wall is replaced by scar tissues.

Fig. 44.—Common duct almost always has stones.

Fig. 45.—The liver is grossly cirrhotic; function is deranged.

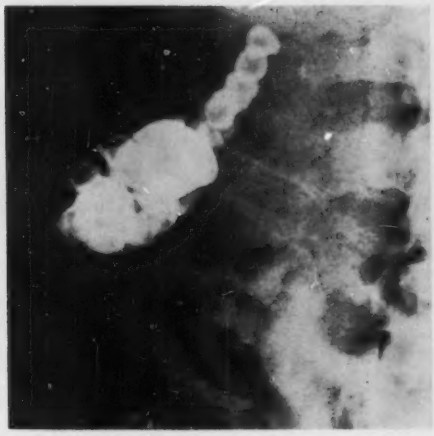


Fig. 46.—No function or visualization of the gallbladder on oral cholecystography.

Stage IV. Not Always Associated with Stones but Actually the Most Advanced Form of the Disease: Found in 3% of Surgical Patients

Fig. 47.—Carcinoma of the gallbladder. Over 90% of women with carcinoma of the gallbladder also had stones.

Fig. 48.—Cholecystoduodenal fistula.

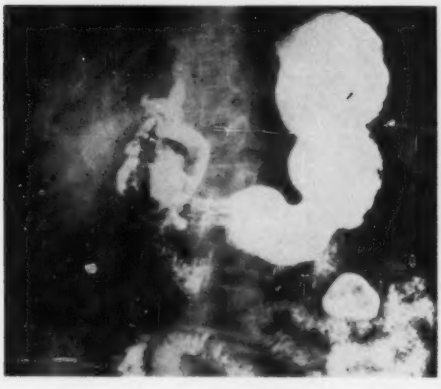


Fig. 49.—Cholecystoduodenal fistula outlined by barium.

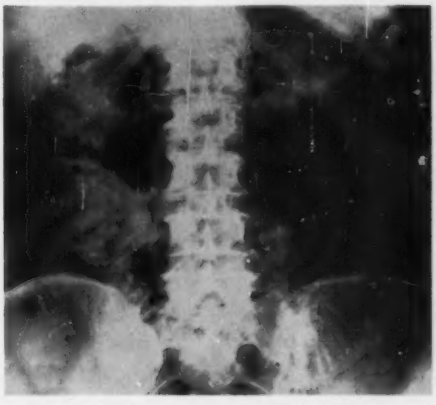


Fig. 50.—The three signs of gallstone ileus. *A*, air in the biliary tree. *B*, small bowel obstruction. *C*, gallstone.

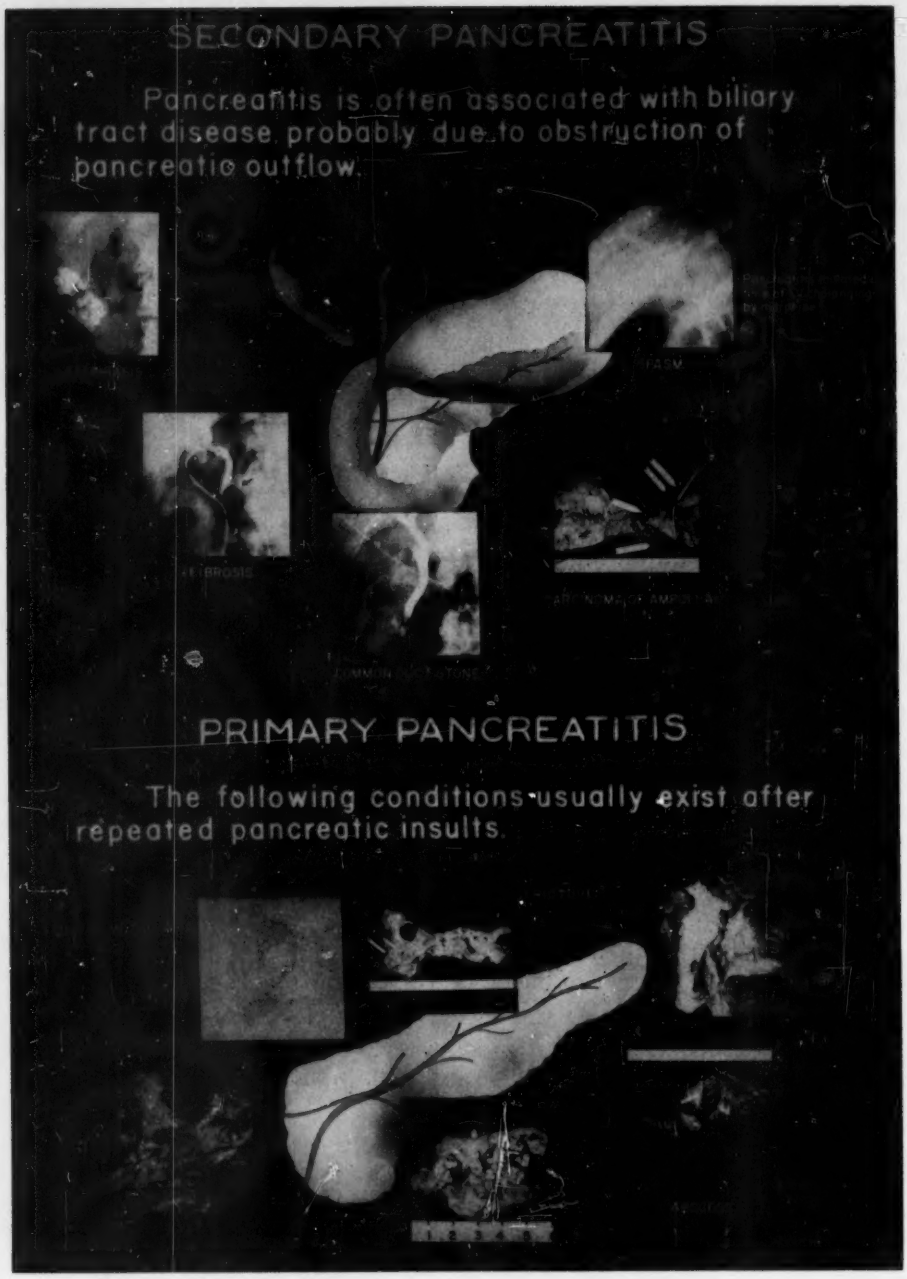

SECONDARY PANCREATITIS
Pancreatitis is often associated with biliary tract disease probably due to obstruction of pancreatic outflow.
PRIMARY PANCREATITIS
The following conditions usually exist after repeated pancreatic insults.

Figure 1 of facing article.

Surgical Management of Chronic Pancreatitis*

L. D. HILL, M.D.; C. S. STONE Jr., M.D.; J. W. BAKER, M.D.; R. F. JONES, M.D.; C. C. PEARSON, M.D.; R. CLEMENTS, M.D.; H. JONES, M.D., and RALPH ODELL, M.D., Seattle

The purpose of this exhibit is to present the practical aspects of the etiologic mechanisms and the surgical management of this disease.

Pancreatitis is a disease of multiple causes.

Repeated episodes of pancreatitis, edema, and necrosis lead to chronic pancreatitis.

Pancreatic ductal obstruction exists in most instances.

The majority of cases are associated with biliary tract disease and/or alcoholism.

In some patients no causative factor can be found.

Alcohol

The incidence of alcoholism in patients with pancreatitis is high. Alcohol stimulates pancreatic flow and at the same time causes pancreatic ductal obstruction.

Other Causes

Other causes of pancreatitis are as follows:

- Trauma
- Metabolic disturbances
- Heredity
- Toxic conditions
- Vascular
- Infection

Surgical Management
Selection of Patients

Indications
- Demonstrable biliary tract disease
- Intractable pain
- Duodenal loop changes with or without obstruction
- Questionable carcinoma
- Progressive pancreatic insufficiency
- Pancreatic calcinosis

Contraindications
- Continuing alcoholism
- Continuing drug addiction

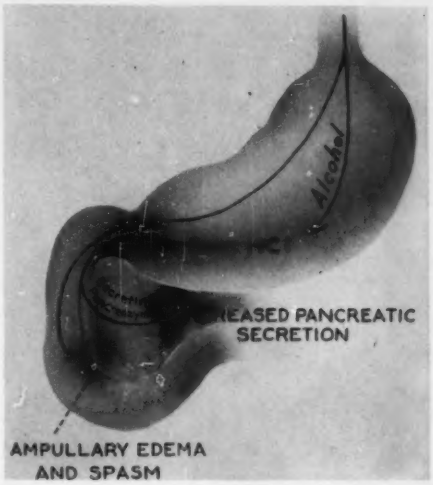


Figure 2

Surgical procedures must be altered to meet the varied causes of pancreatitis. No single operation is suitable for all cases.

For those patients with biliary tract or extrapancreatic pathology and secondary pancreatitis, indirect operations are used. For primary pancreatitis with ductal obstruction, a direct operation on the pancreas is essential.

In secondary pancreatitis, we have used the following procedures:

1. Cholecystectomy, cholangiogram, and pancreatogram
2. Choledochostomy, dilatation of the sphincter of Oddi
3. Transduodenal sphincterotomy

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Present address of Dr. H. Jones and Dr. Odell, Medford, Ore.

* Surgery Illustrated. Shown as a scientific exhibit of the Section on General Practice, at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

4. Excision, cystic duct remnant
5. Exploration, duct of Wirsung and duct of Santorini
6. Vagotomy
7. Subtotal gastrectomy
8. Gastrojejunostomy
9. Choledochojejunostomy, Roux-Y

These procedures will suffice in early, milder forms of pancreatitis, but will often fail with intrapancreatic ductal obstruction.

A Practial Surgical Plan for Secondary Pancreatitis

Remove biliary tract pathology and observe the pancreas. If chronic pancreatitis is present, obtain a reflux pancreatogram to demonstrate patency or obstruction of the pancreatic duct (Fig. 3).

Figure 3

When the pancreatic duct does not fill, in the presence of demonstrable chronic pancreatitis, and the sphincter of Oddi is fibrosed, a sphincterotomy is done (Fig. 4, color plate).

The pancreatic duct should be explored at time of sphincterotomy by direct pancreatogram (Fig. 5) or by cannulation of the duct of Wirsung. These procedures are hazardous, and must be done without pressure or trauma.

When pancreatic ductal obstruction is present a direct operation on the pancreas is necessary.

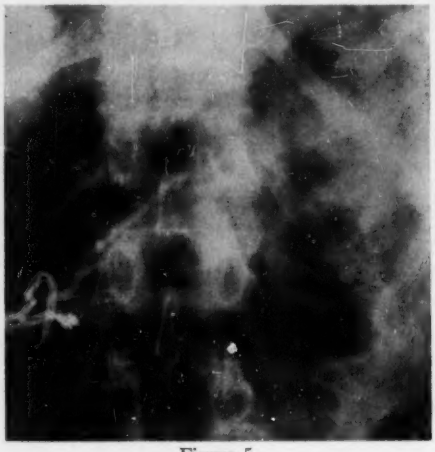


Figure 5

Results of Indirect Procedures

Patients	18		Operations		30
Good	10	Fair	5	Poor	3

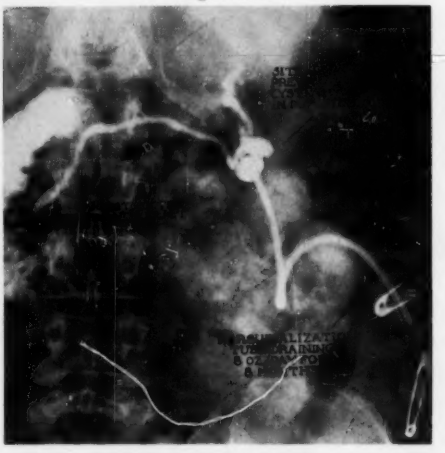
(2, Alcoholism, 1, Hemochromatosis)

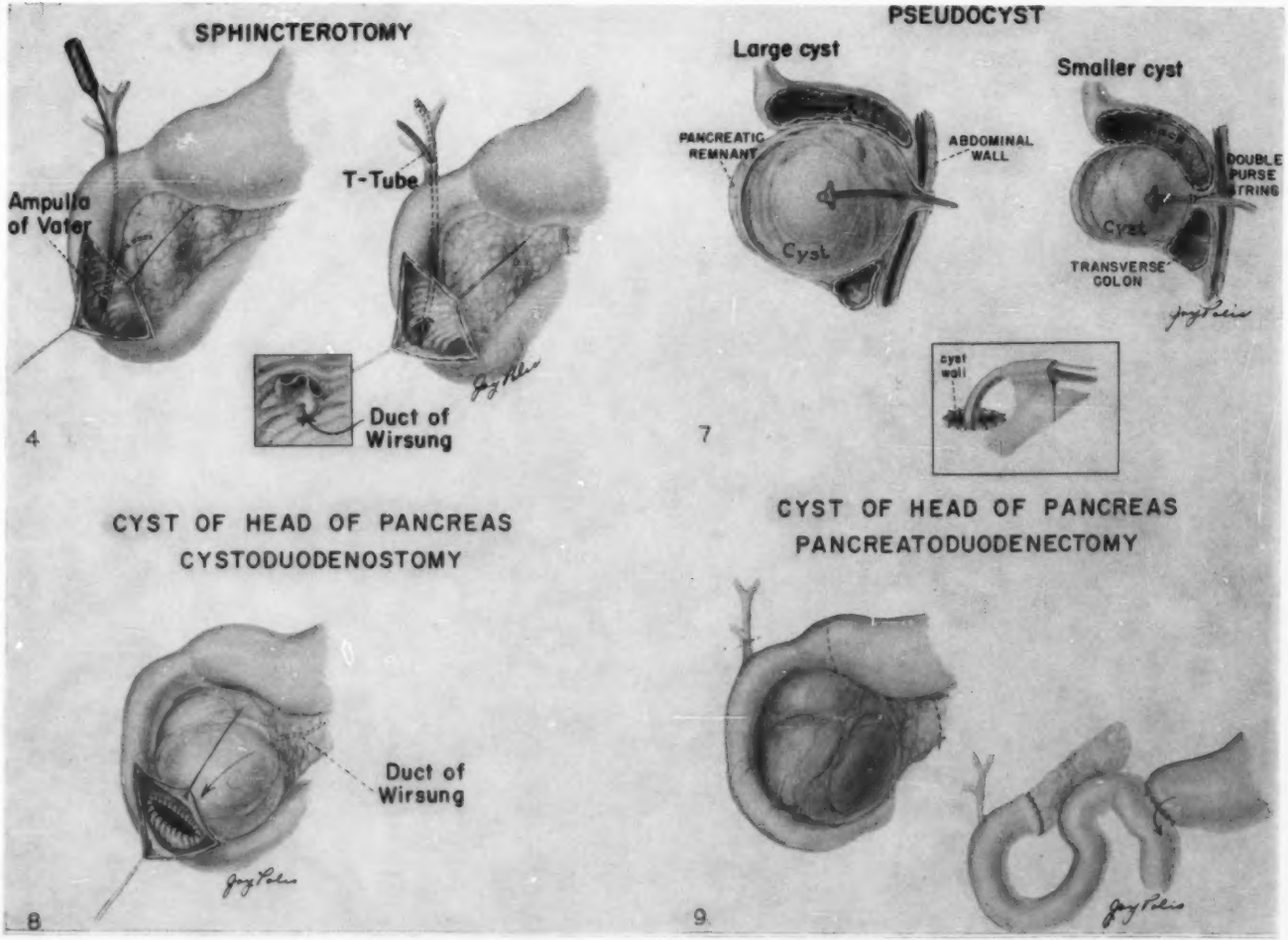
Direct Procedures for Primary Pancreatitis

Primary pancreatitis is manifest by cyst formation, fibrosis, and calcinosis. All of these may be present in the pancreas simultaneously.

Figure 6 shows communication of cyst with duct of Wirsung. Cyst cleared after eight months of drainage by marsupialization with a de Pezzer catheter.

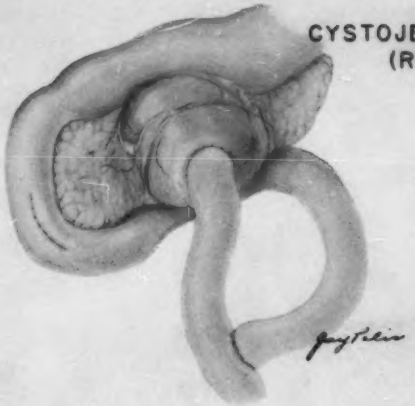
Figure 6




SPHINCTEROTOMY
Ampulla of Vater
T-Tube
Duct of Wirsung
4
PSEUDOCYST
Large cyst
Smaller cyst
PANCREATIC REMNANT
ABDOMINAL WALL
DOUBLE PURSE STRING
Cyst
Cyst
TRANSVERSE COLON
cyst wall
7
CYST OF HEAD OF PANCREAS
CYSTODUODENOSTOMY
CYST OF HEAD OF PANCREAS
PANCREATODUODENECTOMY
Duct of Wirsung
8
9

CYST OF BODY OF PANCREAS

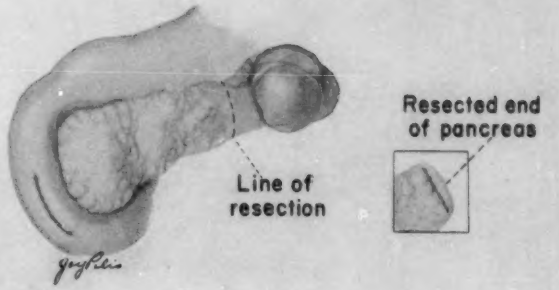
CYSTOJEJUNOSTOMY
(ROUX-Y)



10

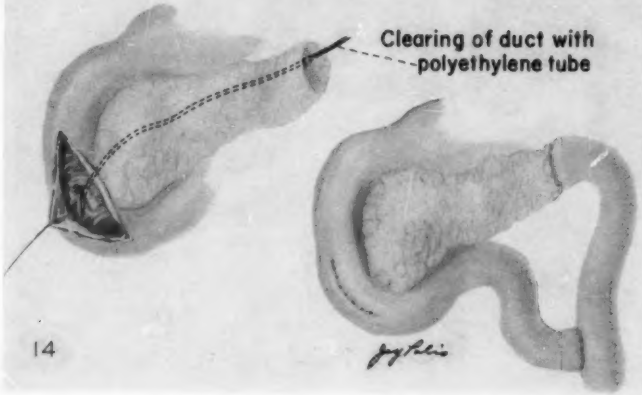
CYST OF TAIL OF PANCREAS

DISTAL PANCREATECTOMY



11

DISTAL PANCREATECTOMY WITH
PANCREATOJEJUNOSTOMY



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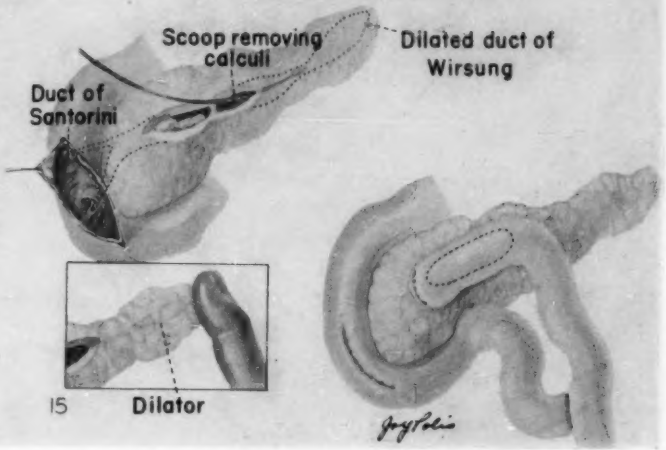
PANCREATOJEJUNOSTOMY



15

For pseudocysts, marsupialization is often the only procedure feasible (Fig. 7, Color Plate).

Cysts of the Head of the Pancreas

For cysts of the head of the pancreas, cystoduodenostomy is the preferred method in our hands. Three patients are alive and well eight, three, and two years after cystoduodenostomy (Fig. 8, Color Plate).

Pancreatoduodenectomy is an alternate procedure for cysts of the head of the pancreas (Fig. 9, Color Plate).

Cysts of the Body of the Pancreas

A cyst of the body of the pancreas, treated by cystojejunostomy (Roux-Y) is shown in Figure 10 (Color Plate).

Cysts of the Tail of the Pancreas

A cyst of the tail of the pancreas, with distal pancreatectomy is shown in Figure 11 (Color Plate).

Fibrosis and Calcinosis Without Cyst

These cases present special problems. Fibrosis of the head of the pancreas is difficult if not impossible to differentiate from carcinoma and often has obstructed the common bile duct and the duct of Wirsung.

Fibrosis and calcinosis chiefly involving the tail of the pancreas—treated by distal pancreatectomy and pancreatojejunostomy—is shown in Figure 14 (Color Plate). The duct of Wirsung was found to be unobstructed by passage of tube.

For diffuse fibrosis and calcinosis with multiple points of ductal obstruction, the duct of Wirsung is opened over a wide area and cleared by scoops and dilated where obstruction is met. A Roux-Y pancreatojejunostomy resulted in satisfactory internal drainage with alleviation of symptoms and improvement in pancreatic function (Fig. 15, Color Plate).

Summary

Chronic pancreatitis is a disease of multiple causes occurring after repeated acute attacks and is associated with ductal obstruction.

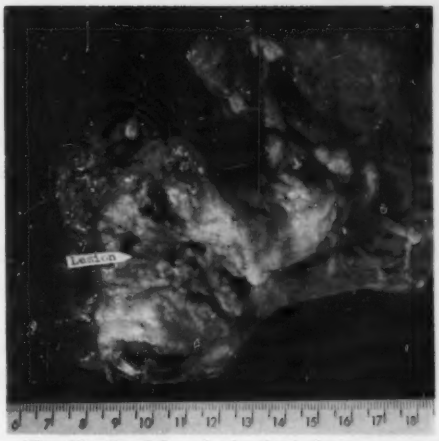


Fig. 12.—Involving the head of the pancreas.

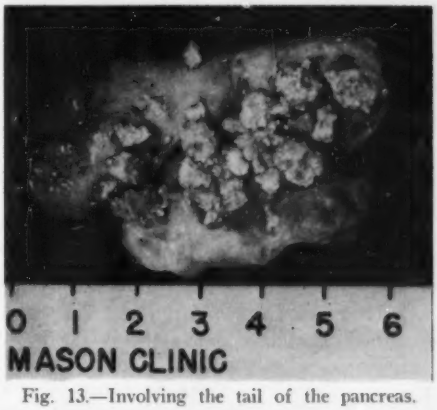


Fig. 13.—Involving the tail of the pancreas.

Better understanding of the etiology has led to more rational medical management.

Surgery is reserved for intractable cases.

At operation, biliary tract pathology should be corrected. This is often sufficient therapy in early secondary pancreatitis.

In the presence of pancreatic ductal obstruction a direct attack on the pancreas is necessary to relieve the patient.

Our preference at the present time is adequate internal drainage with preservation of pancreatic function whenever feasible. Resection is reserved for difficult cases and when carcinoma cannot be ruled out.

A practical plan of surgical management is presented.

Cancer of the Thyroid*

Morphological and Clinical Study of Two Hundred Six Cases

V. E. CHESKY, M.D.; C. A. HELLWIG, M.D.; E. N. McCUSKER, M.D., and J. W. WELCH, M.D., Halstead, Kan.

Terminology

Our terminology incorporates all types of thyroid cancer listed in American and European classifications. The terms used in the different thyroid clinics have been correlated.

TABLE 1.—*Classification*

Wegelin	Frazell-Foote	Warren-Meissner	Hertzler Clinic
1. Metastasizing adenoma		Adenoma with invasion	Invasive adenoma
2. Papilloma	Papillary carcinoma	Papillary carcinoma	Papilloma
3.	Follicular & alveolar ca.	Follicular carcinoma	Follicular carcinoma
4. Large-cell small-alveolar carcinoma	Huerthle-cell carcinoma	Huerthle cell ca.	Huerthle-cell tumor
5. Parastruma			Clear-cell tumor
6. Langhans' wuchernde struma			Langhans' wuchernde struma
7. Carcinoma solidum	Solid adenocarcinoma	Carcinoma simplex	Solid large-cell carcinoma
8. Columnar-cell carcinoma			Columnar-cell carcinoma
9. Squamous-cell carcinoma	Squamous carcinoma	Epidermoid carcinoma	Squamous-cell carcinoma
10. Spindle-cell sarcoma	Fibrosarcoma	Fibrosarcoma	Spindle-cell tumor
11. Polymorph-cell sarcoma	Giant-cell carcinoma	Giant-cell carcinoma	Polymorph-cell tumor
12. Round-cell sarcoma		Small-cell carcinoma	Round-cell tumor
		Lymphoma	
13. Hemangioendothelioma			Hemangioendothelioma

Geographic Pathology

The incidence of the different goiter types is given for American and European localities. Papillary carcinoma is the commonest type in America, but rare in Switzerland. Proliferant adenoma is the commonest type in Switzerland, but almost unknown in America.

TABLE 2.—*Geographic Pathology of Malignant Goiter*

	Berne (1950), %	Boston (1936), %	Halstead (1955), %	206 Cases
Invasive adenoma	2	25	22.3	46
Papillary carcinoma	18	35	31.1	64
Follicular carcinoma		12	6.3	13
Huerthle-cell tumor	22	2	10.7	25
Clear-cell tumor			3	6
Langhans' wuchernde struma	32		3.9	8
Solid large-cell carcinoma			3.9	8
Columnar-cell carcinoma			3.4	7
Squamous-cell carcinoma		0.5	1	2
Spindle-cell carcinoma		2	1.9	4
Polymorph-cell carcinoma		8.2	6.4	13
Round-cell carcinoma		16.4	4.4	9
Hemangioendothelioma	16		1	2
Differentiated:	74	76	78.3	161 cases
Undifferentiated:	26	24	21.7	45 cases

Submitted for publication Aug. 6, 1959.

From the Hertzler Clinic and Hertzler Research Foundation.

Aided by a grant of the American Cancer Society, Kansas Division.

* Surgery Illustrated. Shown as a scientific exhibit of the Section on Pathology and Physiology, at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

Frequency, Age, and Weight

Our 206 cases were found in 3,029 goiter operations, giving a percentage of 6.8; 13.8% of the solitary adenomas were malignant, but only 1.1% of diffuse goiters.

The average age was lower in the less malignant types than in highly malignant goiters (45 vs. 56 years).

The weight of the surgical specimens was three times as much in highly malignant goiters as in less malignant (140 vs. 53 gm.).

TABLE 3.—*Relative Frequency of Cancer of the Thyroid*

In 20 years....with 3,029 thyroid operations: 206 cases of cancer of the thyroid 6.8 %		
1,908 nodular goiters	1,121 diffuse goiters	1,030 solitary adenomas
194 malignancies	12 malignancies	142 malignancies
▼	▼	▼
9.1 %	1.1 %	13.8 %

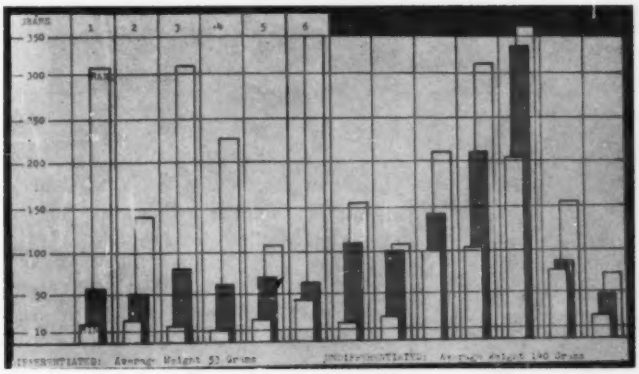


Fig. 1.—Weight of operative specimens.

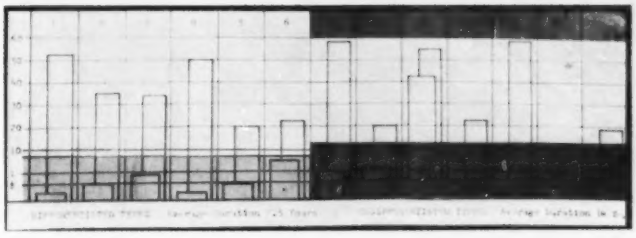


Fig. 2.—Duration of goiter.

Preoperative Diagnosis

Cancer was suspected in 14.3% of thyroid cancer cases.

TABLE 4.—*Clinical Diagnosis*

Type of Cancer	1	2	3	4	5	6	7	8	9	10	11	12	13
Solitary adenoma	36	31	5	10	4	3	4	5	1	1	7	4	2
Diffuse toxic goiter	2	5	3	2									
Nodular toxic goiter	1	4	2	7									
Nodular colloid goiter	5	8	1	4	2	4		3		2	2		
Thyroiditis												1	
Cancer	2	9	1	2		1	4	1	1	1	4	4	

Differentiated: Cancer diagnosed in 9.3% Undifferentiated: 31.1%
All cases: 29 of 206 correctly diagnosed: 14.3%

TABLE 5.—*Metastases*

Type of Cancer	1	2	3	4	5	6	7	8	9	10	11	12	13
Cervical lymph nodes		6	2	1	1							1	
Mediastinum					1							1	
Lung		1			1		2					1	
Bone		3	3										
Other distant organs		1										1	
Invasion neck organs			1		1			5	1	1	4	4	

TABLE 6.—*Previous Operations*

Type of Cancer	1	2	3	4	5	6	7	8	9	10	11	12	13
		3		3			2	1			1	0	1

6 of differentiated (3.7%) — 4 of undifferentiated (9%)

10 of all — 206 cancer cases (5%)

Metastases

Clinical evidence of metastases was more frequently present in less malignant tumors. The highly malignant thyroid cancers killed the patient by local extension to vital neck organs before distant metastases had a chance to cause clinical symptoms.

TABLE 7.—*Complaints*

Type of Cancer	1	2	3	4	5	6	7	8	9	10	11	12	13
Swelling in Neck	42	55	12	25	4	7	7	7	2	3	13	7	2
Rapid increase	3	2	3	1		3	2	1		1	4		1
Pressure	2												
Hoarseness	3			2					1	1			
Dysphagia									1	2			
Dyspnea		9	3	2		3	2	1	1	1	5	1	
Choking	4	4	3				1			2	3		
Cough	1						1				2		
Weight loss		18		8		4	2	2		1	5	3	1
Pain													
Weakness	4			2									
Rapid heart	10	10	3	9	3								
Nervousness	8	11			4								

Characteristics

Eighteen photographs illustrate the clinical, gross, and microscopic characteristics of differentiated and undifferentiated tumors of the thyroid. Thirteen types with different morphological and clinical features are distinguished.

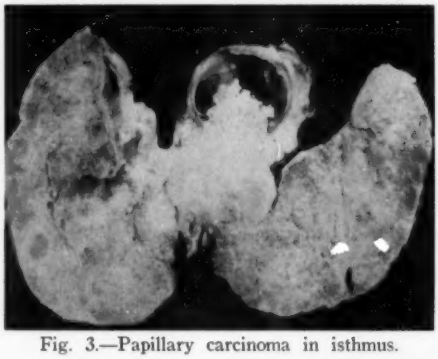


Fig. 3.—Papillary carcinoma in isthmus.

Fig. 4.—Adolescent with papillary carcinoma.

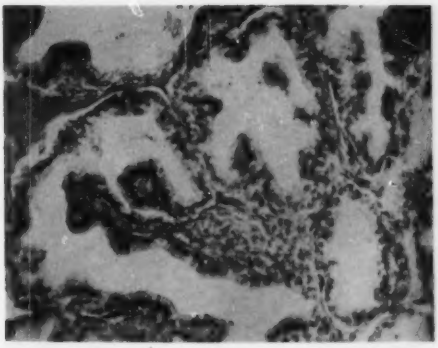


Fig. 5.—Pure papillary carcinoma.

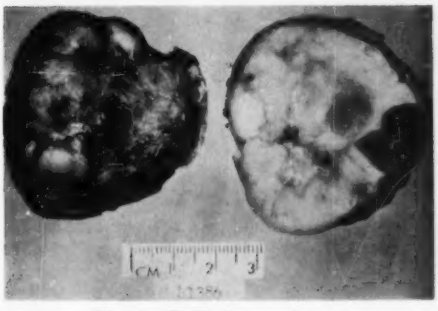


Fig. 6.—Follicular carcinoma.

Fig. 7.—Metastases of follicular carcinoma in frontal bone.

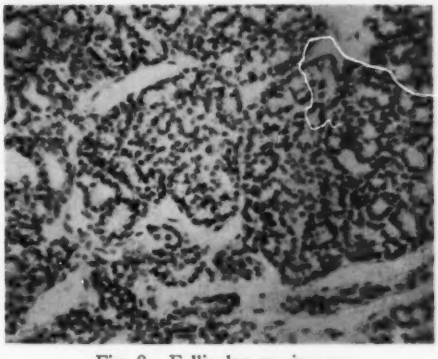


Fig. 8.—Follicular carcinoma.

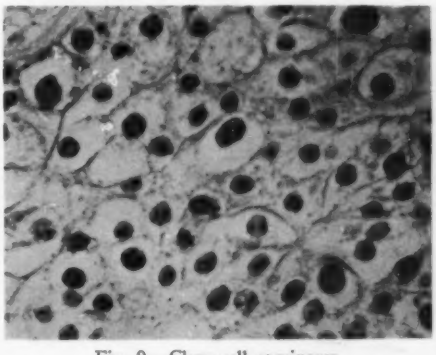


Fig. 9.—Clear-cell carcinoma.

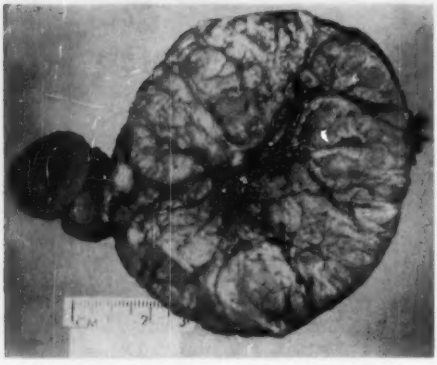


Fig. 10.—Wuchernde struma of Langhans.

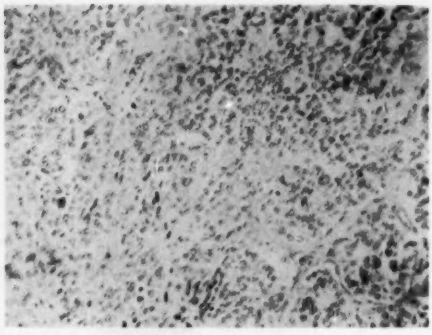


Fig. 11.—Wuchernde struma of Langhans.

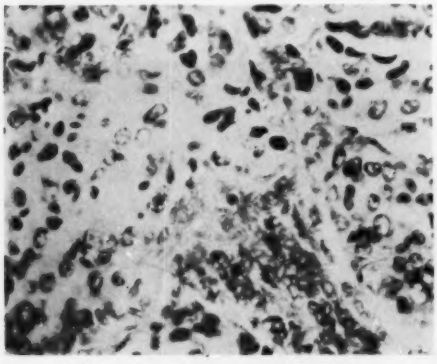


Fig. 12.—Large-cell carcinoma.

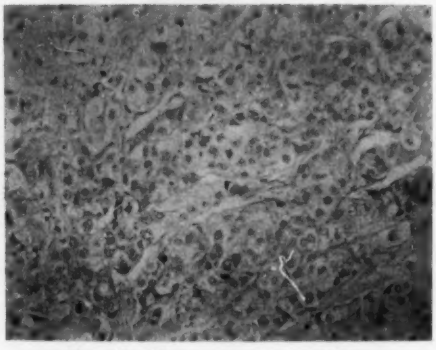


Fig. 13.—Squamous-cell carcinoma.

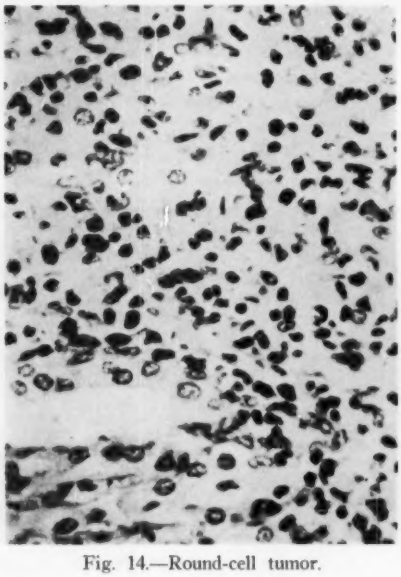


Fig. 14.—Round-cell tumor.

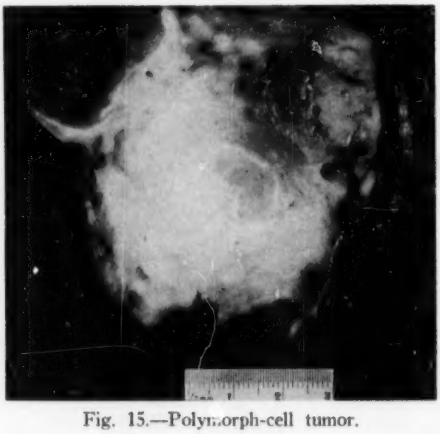


Fig. 15.—Polymorph-cell tumor.

Fig. 16.—Recurrent polymorph-cell tumor in 75-year-old patient.

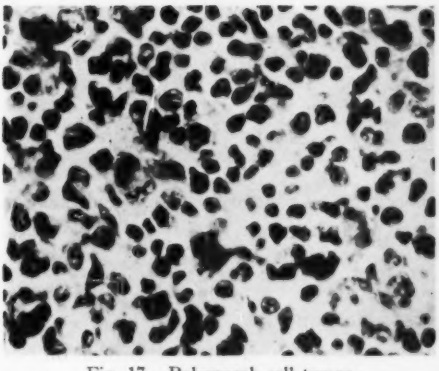


Fig. 17.—Polymorph-cell tumor.

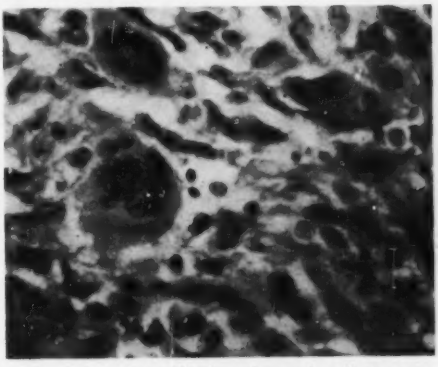


Fig. 18.—Giant cells in polymorph-cell tumor.

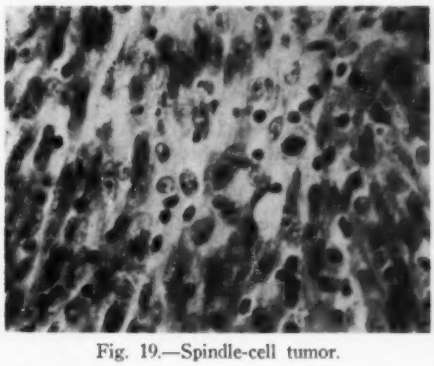


Fig. 19.—Spindle-cell tumor.

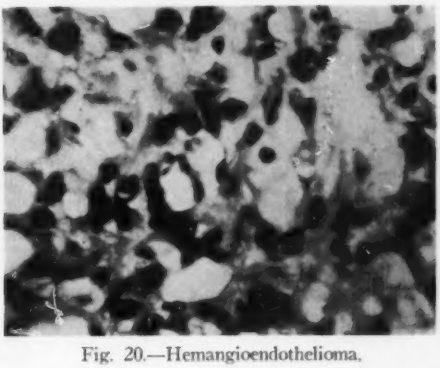


Fig. 20.—Hemangioendothelioma.

Treatment

In low malignancy, lobectomy or bilateral subtotal thyroidectomy was the method of choice. Cervical lymph nodes were dissected only when clinically enlarged. The results in this group of tumors (about 80% of the total) were excellent.

TABLE 8.—*Treatment*

Type of Cancer	1	2	3	4	5	6	7	8	9	10	11	12	13	206 Cases %
Lobectomy	29	34	6	13	2	4	7	5	2	1	9	2	2	56.3
Subtotal thyroidectomy	17	24	6	11	4	4	1	2		2	2	2		36.4
Total thyroidectomy			1				1			2	1			2.4
Radical neck dissection		4	1											2.4
Cervical lymph nodes		3		1	1									2.4
Tracheotomy											1	4	4	4.4
X-ray therapy	3	5									1		2	7.3
Radioiodine treatment		2	1											1.5

TABLE 9.—*Follow-Up Observations*

Type of Cancer	1	2	3	4	5	6	7	8	9	10	11	12	13
Living and well	44	43	8	18	3	5	1	2		1		2	1
Living with disease		7	1				1						
Recurrence		8	1	4	1		6	1					1
Reoperated		4		2	1								
Died of cancer		4	3		2	1	6	2	2	3	13	7	
Died of other causes	2	1		3									
Lost during follow-up	2	6	1			2		3					1
Five-year survival	96%	88%	77%	75%	50%	63%	13%	29%	0	25%	0	22%	50%

Differentiated: 81% Five-year survival Undifferentiated: 17% Five-year survival

A More Optimistic Approach to Cancer of the Thyroid

A diagnosis of malignant goiter without further qualification is meaningless, because the grade of malignancy of thyroid cancer may vary from an excellent prognosis after conservative surgery to complete hopelessness in spite of the most radical therapeutic measures.

Eighty per cent of thyroid cancers have a slow course and can be treated with lobectomy, while twenty per cent have a very poor prognosis whatever the treatment.

The prognosis of a given case depends, more than on any other factor, on its microscopic structure.

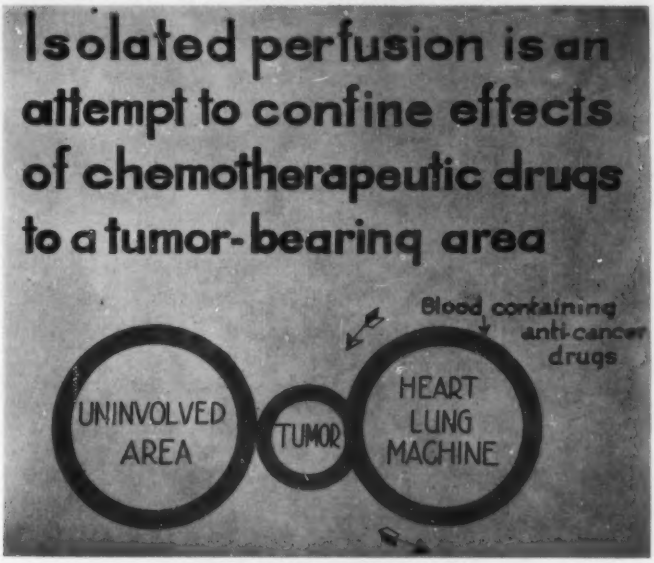
Therefore a detailed classification of malignant goiter is imperative and of greatest practical importance.

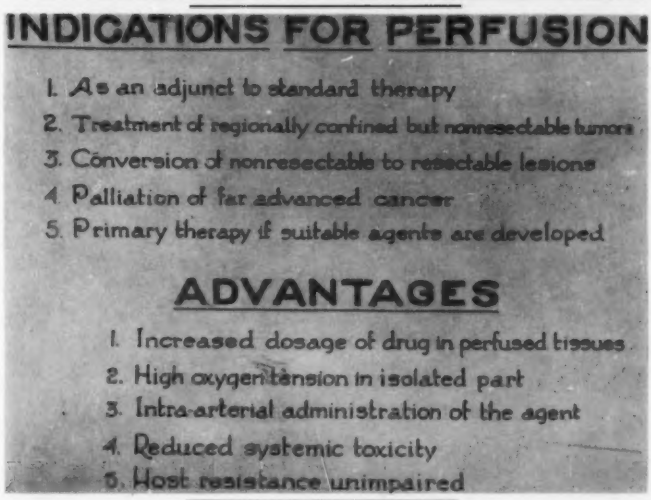
327 Chestnut St.

The Treatment of Cancer by Perfusion*

OSCAR CREECH Jr., M.D.; E. T. KREMENTZ, M.D.; R. F. RYAN, M.D.; KEITH REEMTSMA, M.D.; J. N. WINBLAD, M.D., and JAMES L. ELLIOTT, M.D., New Orleans

Technique


Isolated perfusion is an attempt to confine effects of chemotherapeutic drugs to a tumor-bearing area
Blood containing anti-cancer drugs
UNINVOLVED AREA
TUMOR
HEART LUNG MACHINE


INDICATIONS FOR PERFUSION
1. As an adjunct to standard therapy
2. Treatment of regionally confined but nonresectable tumors
3. Conversion of nonresectable to resectable lesions
4. Palliation of far advanced cancer
5. Primary therapy if suitable agents are developed
ADVANTAGES
1. Increased dosage of drug in perfused tissues
2. High oxygen tension in isolated part
3. Intra-arterial administration of the agent
4. Reduced systemic toxicity
5. Host resistance unimpaired

Received for publication Aug. 14, 1959.

Tulane University School of Medicine; Charity Hospital of Louisiana; Touro Infirmary; U.S. Public Health Service Hospital.

* Surgery Illustrated. Shown as a scientific exhibit of the Section on Surgery, General and Abdominal, at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

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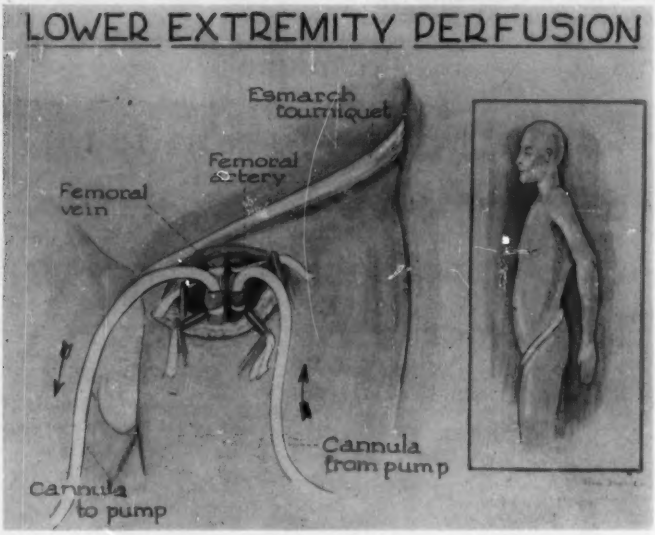


Figure 3

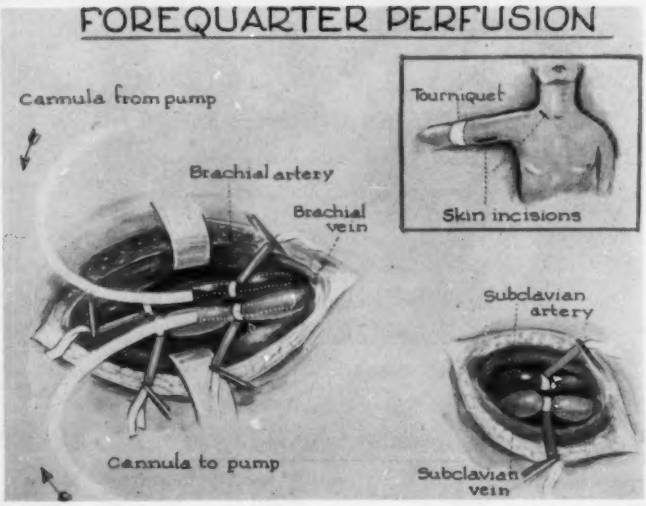


Figure 4

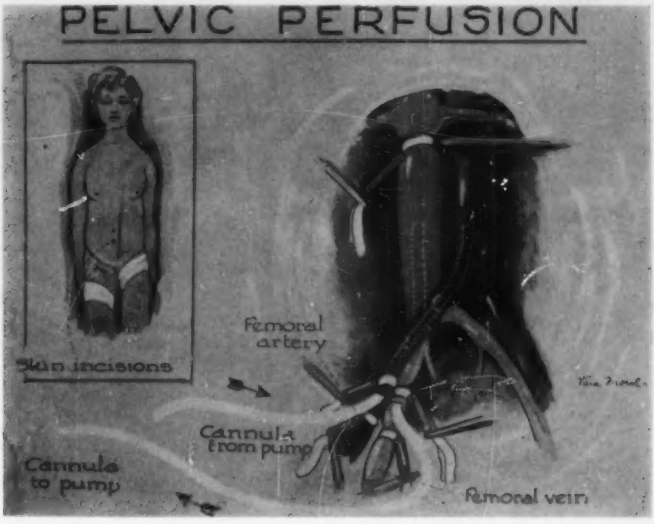


Figure 5

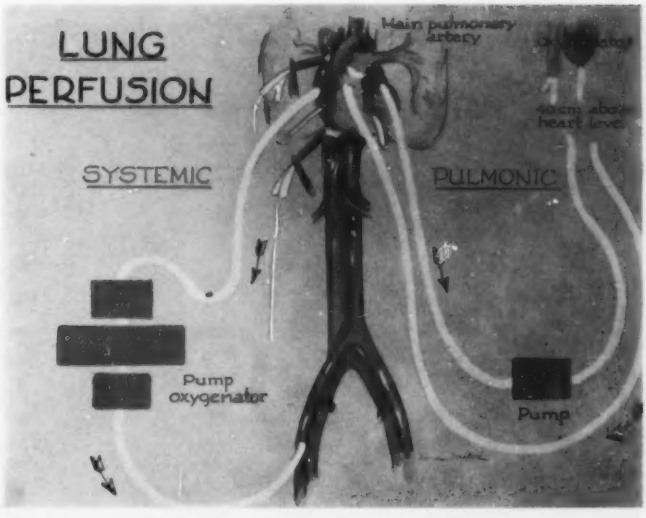


Figure 6

Technique

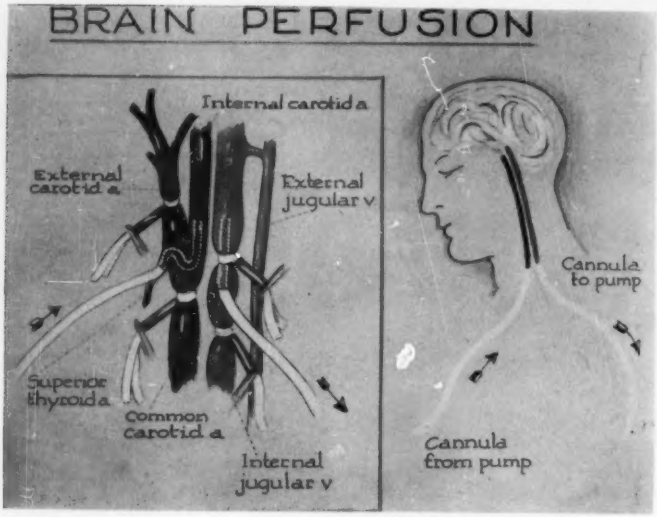


Figure 7

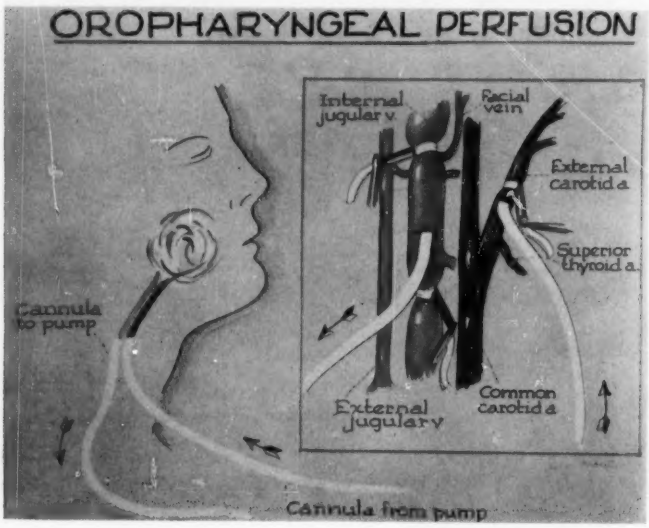


Figure 8

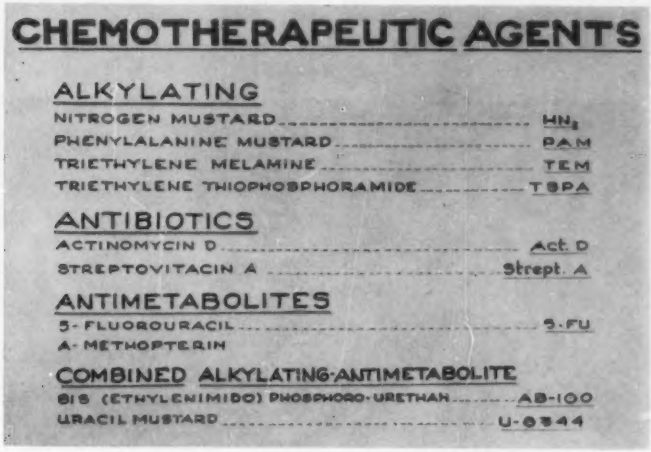


Figure 9

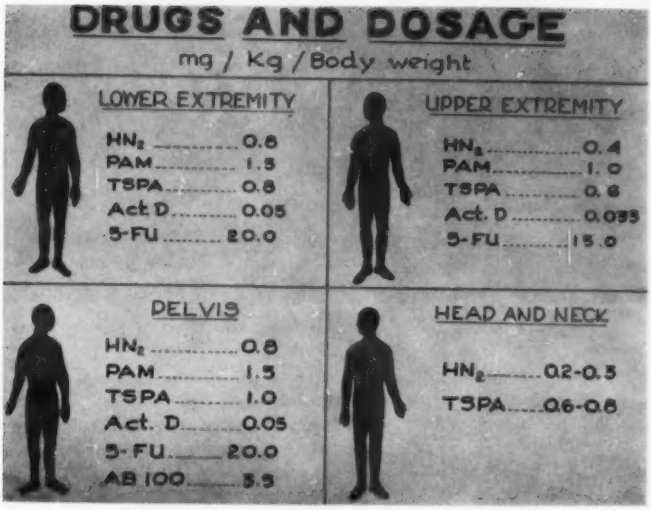


Figure 10

Metastatic Melanoma

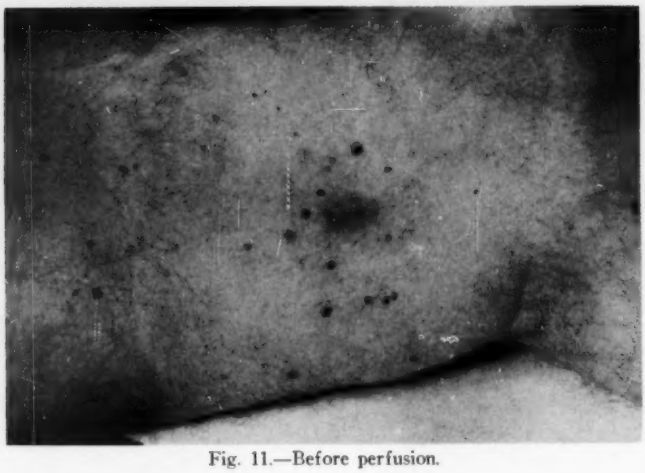


Fig. 11.—Before perfusion.

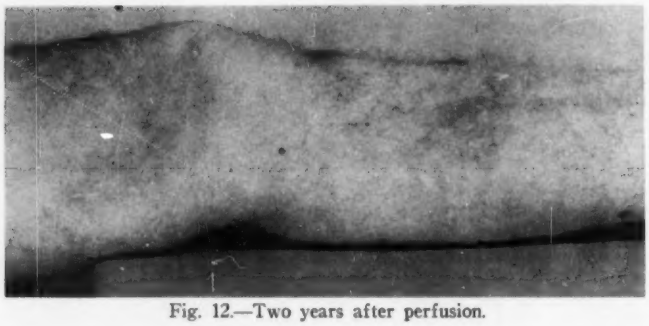


Fig. 12.—Two years after perfusion.

CASE 1.—A 77-year-old white man. In 1956 malignant melanoma removed from left foot, followed by groin dissection May, 1957: Appearance of multiple dermal metastases. June, 1957: Isolated perfusion with 120 mg. phenylalanine mustard (PAM) through common femoral artery.

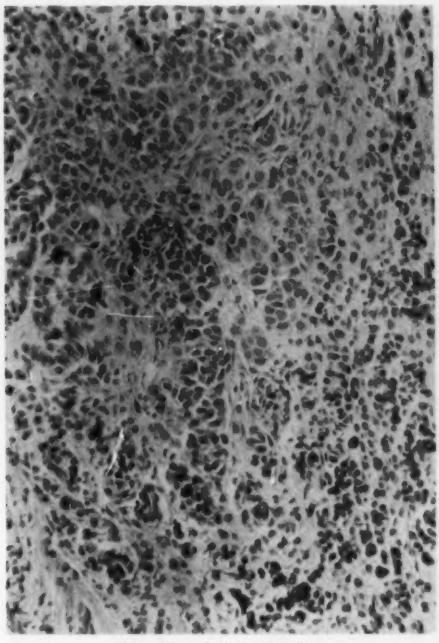


Fig. 13.—Original lesion.

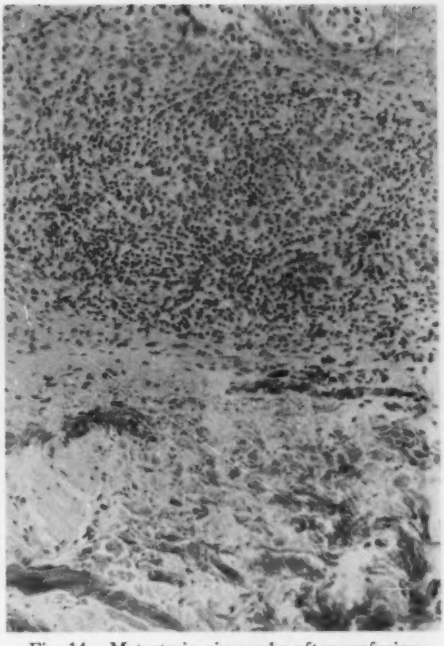


Fig. 14.—Metastasis, six weeks after perfusion.

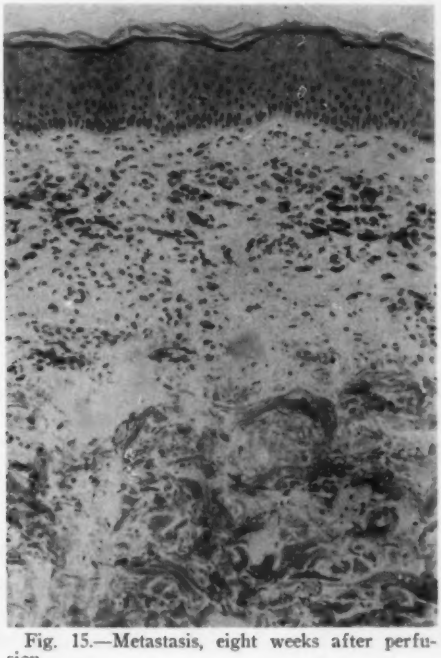


Fig. 15.—Metastasis, eight weeks after perfusion.

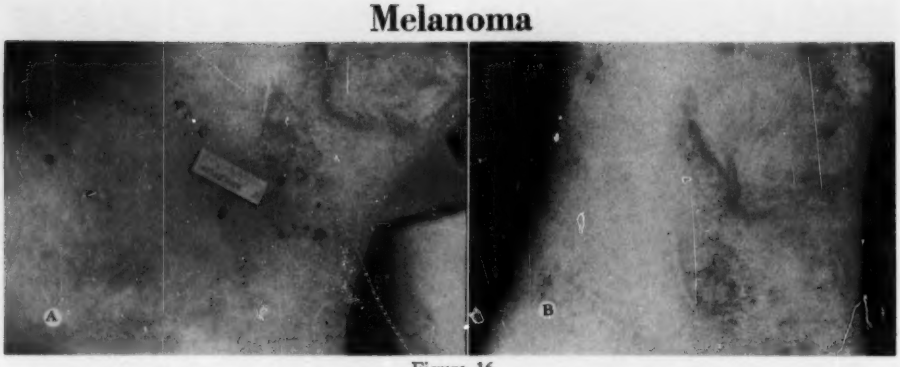


Figure 16

CASE 2.—1947: Small mole (malignant melanoma) removed from right medial lower leg. 1958: Local recurrence followed by multiple excisions. Treated with TSPA intra-arterially without improvement (Fig. 16, *A*). December, 1958: Common femoral artery perfusion with 130 mg. PAM (1.8 mg. per kilogram of body weight). January, 1959: Many lesions disappeared (Fig. 16, *B*).

Figure 17

CASE 3.—1957: Malignant melanoma with extensive metastases developed on right leg. Repeated x-ray therapy without response (Fig. 17, *A*). December, 1958: Common femoral artery perfusion with PAM, 123 mg. (1.5 mg. per kilogram of body weight) and triethylene thiophosphoramide (TSPA), 65 mg. (0.8 mg. per kilogram of body weight). May, 1959: Residual disease continues to regress (Fig. 17, *B*).

Carcinoma

CASE 4.—1946: Hysterectomy for endometrial adenocarcinoma. 1955: Metastasis discovered in ischium. Treated with excision, x-ray therapy, testosterone, and TSPA. Disease continues to progress (Fig. 19). August, 1957: Chordotomy for relief of pain. January, 1958: Abdominal exploration; lesion nonresectable. Pelvic perfusion, HN_2, 20 mg. (0.4 mg. per kilogram of body weight [Figs. 18, 20]). March, 1958: Tumor necrotic, liquefied, and drained. June, 1958: Persistent disease apparent. Hind quarter amputation performed (Fig. 21). June, 1959: No evidence of recurrence (Fig. 22).

Figure 18

Figure 19

Figure 20

Figure 21

Figure 22

Sarcoma

CASE 5.—1941: Myxosarcoma of left leg excised. 1957: Recurrence of tumor. Patient refused amputation (Fig. 23, *A*). February, 1958: Common femoral artery perfusion with PAM, 138 mg. (2 mg. per kilogram of body weight). March, 1958: Sufficient regression to carry out wide local excision. Tumor largely necrotic or undergoing regressive changes (Fig. 23, *B*). 1959: No evidence of recurrence.

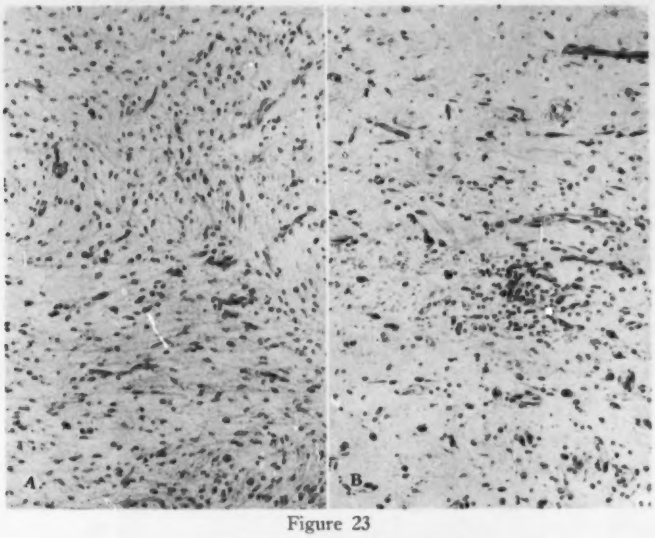


Figure 23

CASE 6.—1957: Fibrosarcoma, plantar surface of right foot, treated by wide excision. April, 1958: Recurrent lesion excised. November, 1958: Metastases to right foot and leg. Local excision and groin dissection (Fig. 24, *A*). December, 1958: Perfusion PAM, 94 mg. (1.5 mg. per kilogram of body weight) and TSPA, 45 mg. (0.8 mg. per kilogram of body weight). May, 1959: Disease regressing (Fig. 24, *B*).

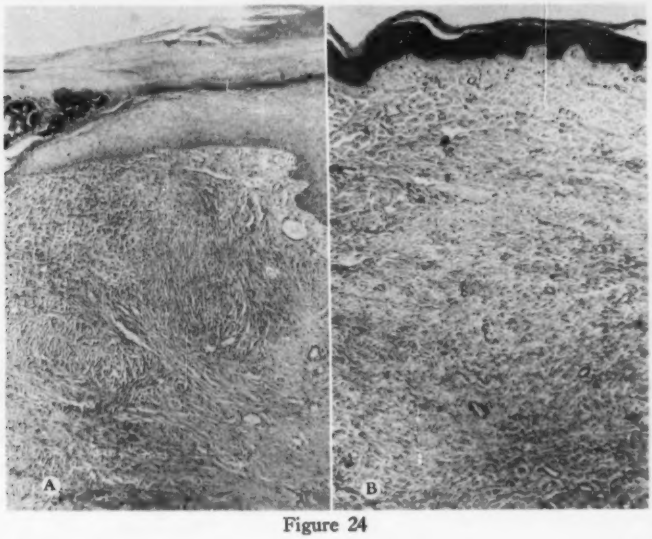


Figure 24

Results

PERFUSIONS

	NUMBER OF PATIENTS	NUMBER OF PERFUSIONS
MELANOMA	36	44
SARCOMA	26	29
CARCINOMA	69	71
GLIOBLASTOMA	5	5
TOTAL	136	149

Figure 25

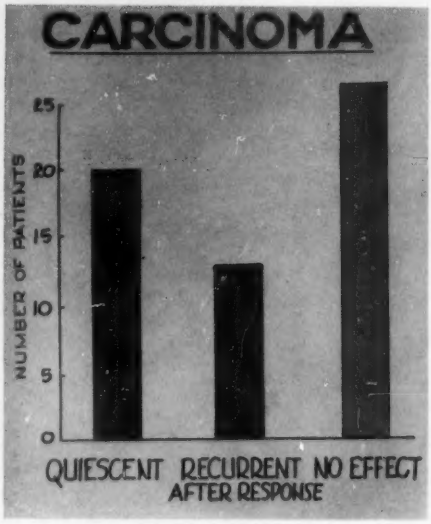


Figure 26

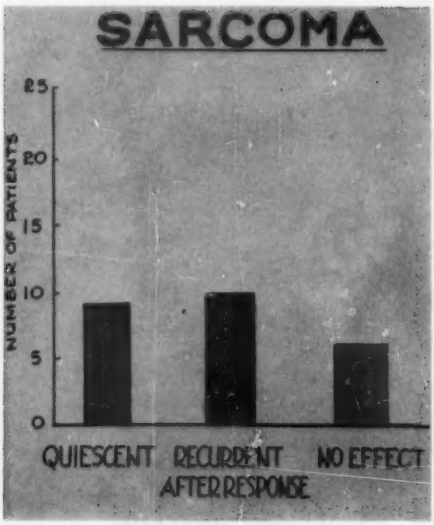


Figure 27

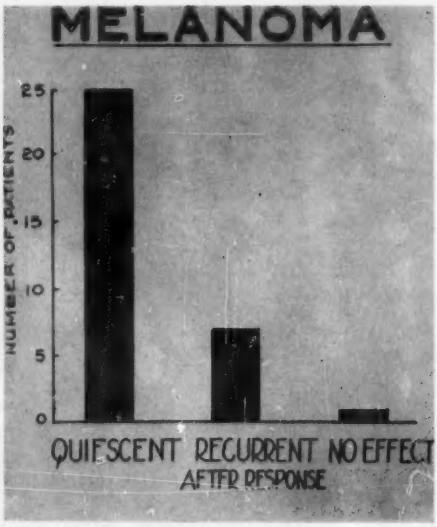


Figure 28

Apparatus

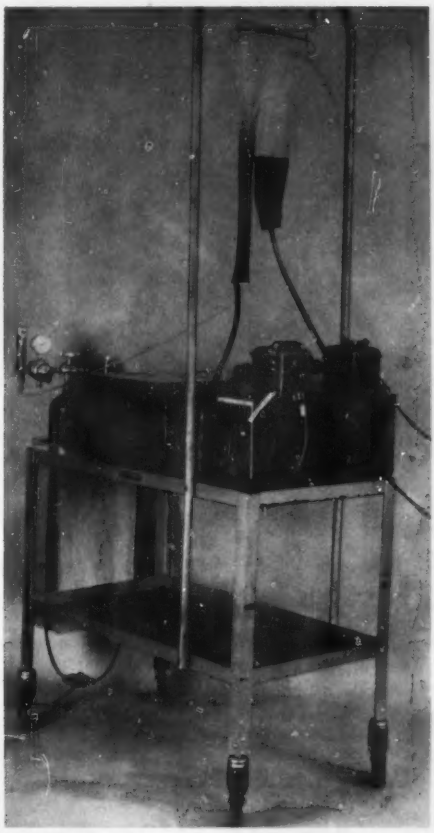


Fig. 29.—Perfusion apparatus consisting of two Sigmamotor pumps (TM5) and bubble type oxygenator (Pulmopak).

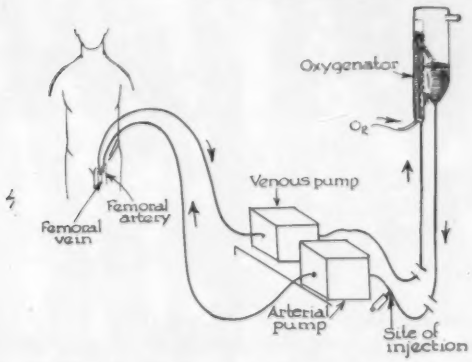


Fig. 30.—Diagram of perfusion apparatus in operation.

Preanesthetic Medication*

Old and New Concepts

JOHN ADRIANI, M.D., and O. HORACE YARBERRY Jr., M.D., New Orleans

Premedication is an old idea. Claude Bernard suggested using morphine before anesthesia to facilitate induction. Misconceptions are prevalent concerning premedication. Often it is ordered empirically without rational basis. This exhibit indicates accepted uses and common misconceptions.

Summary

Premedication is part of the anesthetic. The desired goal is tranquility and amnesia without loss of consciousness. The narcotics combined with belladonna alkaloids still remain the most reliable and versatile agents for premedication.

Why Give Premedication?

Preanesthetic medication is necessary (1) for psychic sedation primarily, (2) to minimize secretions, (3) to fortify impotent anesthetics, and (4) as prophylaxis for suppression of vagal and other autonomic reflex activity.

What Are the Criteria for Selection of Premedication?

Premedication is part of the anesthetic. Drugs and methods must be individualized for a particular patient and technique of anesthesia. The anesthetist is best qualified to prescribe it. It should be his prerogative to select it.

Submitted for publication Aug. 14, 1959.

Department of Anesthesia, Charity Hospital, and the Department of Surgery, Louisiana State University, School of Medicine.

128/976

* Surgery Illustrated. Shown as a scientific exhibit of the Section on Anesthesiology at the 108th Annual Meeting of the American Medical Association, Atlantic City, June 8-12, 1959.

What Drugs Are Used?

Central nervous system depressants are necessary for psychic sedation. The narcotics and hypnotics are most widely used. Belladonna alkaloids suppress secretions. Most satisfactory results are obtained with narcotics (opiates and related drugs) combined with belladonna alkaloids.

Is Omission of Premedication Objectionable?

If apprehension is not relieved, patients are difficult to anesthetize. Psychic trauma, prolonged stormy induction, struggling and anoxia due to obstruction from excessive secretions result. The anesthetic risk is thereby increased.

Are the Narcotics Obsolete as Premedicants?

Despite prejudice against narcotics they remain the most effective agents available. Premedication should relieve anxiety. Narcotics produce euphoria and air of indifference unmatched by other depressant drugs. Also, antagonists are available to combat intolerance or overdosage.

Are Barbiturates as Effective as Narcotics?

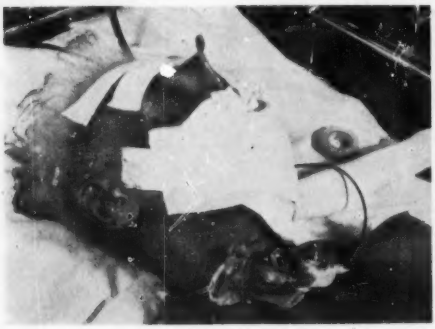
Barbiturates are largely hypnotic. They do not alter the patient's attitude toward his environment as well as the narcotics. Often basal narcotic doses are required. Such doses produce unconsciousness with respiratory and circulatory depression.

How Do Tranquilizers Compare with Narcotics and Hypnotics?

Tranquilizers alone, as a rule, do not provide adequate sedation. They must be used in conjunction with narcotics and hypnotics. Undesirable side-effects, particularly hypotension, are frequently observed.

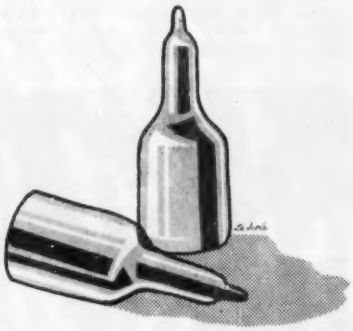
Should Narcotics Be Used Routinely?

A routine is a substitute for thought. Obviously, newborns, small infants, the aged, and patients who are anoxic, in shock, or otherwise acutely ill are intolerant to and should not have narcotics or other depressant drugs.



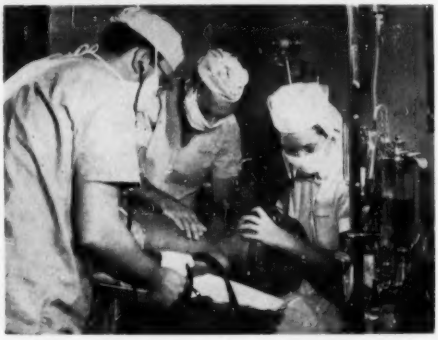
Is Meperidine Preferable to Morphine?

The tranquilizing effect of the synthetic narcotics, such as methadone, meperidine (Demerol), anileridine (Leritine), are adequate but not as effective as morphine or dihydromorphinone (Dilaudid).



What About Phenothiazine Derivatives for Premedication?

This group of drugs, of which chlorpromazine is the prototype, enhances the effects of other central nervous system depressants. They are sympatholytic and ganglioplegic. Thus they can cause hypotension which is difficult to reverse. Promethazine (Phenergan) is the least detrimental.



How Does Advanced Age Modify Choice of Drugs?

Tolerance to narcotics and hypnotics is decreased in the aged. Dosage should be reduced. Barbiturates cause excitement, disorientation, and delirium, and therefore should be omitted.

Are Infants and Children Premedicated in the Same Manner as Adults?

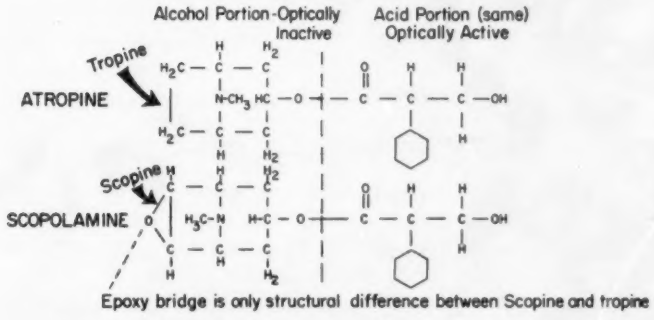
Infants and children respond to narcotics and sedatives in a variable and less predictable manner than adults. Over- or undersedation is common. Barbiturates have a wider margin of safety than narcotics.

What Is the Best Drying Agent?

The belladonna alkaloids are superior to other anticholinergics in providing antisecretory activity and vagal blocking effects with minimal side-effects.

	INCREASE OF PULSE/MIN.	DRYING INDEX
Levo-hyoscyamine (0.5 mg.)	27	14.3
Atropine (0.43 mg.) gr. $\frac{1}{150}$	0	1.5
Atropine (0.65 mg.) gr. $\frac{1}{100}$	8	2.9
Atropine (1.08 mg.) gr. $\frac{1}{60}$	21	5.9
Scopolamine (0.5 mg.)	2	11.1
Dibuline (25 mg.)	32	1.9
Pamine (1 mg.)	58	12.5
Antrenyl (1 mg.)	30	4.6
Dextro-hyoscyamine (0.5 mg.)	0	1.0
Chlortrimetron (10 mg.)	(−) 8	1.2
Saline	0	1.0

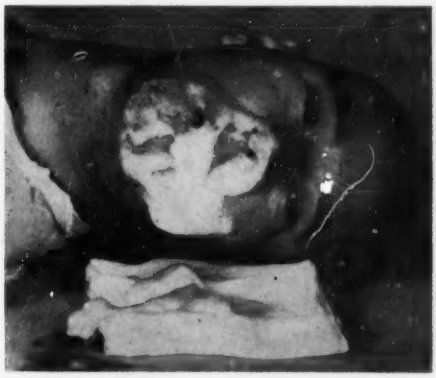
Does Scopolamine Have Advantages over Atropine?



Scopolamine enhances the effects of narcotics and hypnotics and produces amnesia. Also, it has superior drying effects and causes less increase in pulse rate. Atropine accelerates the pulse.

Should Anticholinergics Always Be Used?

Anticholinergics are desirable at all times with all anesthetic techniques for their vagal blocking action, if not for their antisecretory activity. They may be omitted during regional anesthesia when supplementation is not anticipated.



Do the Belladonna Drugs Depress Respiration?

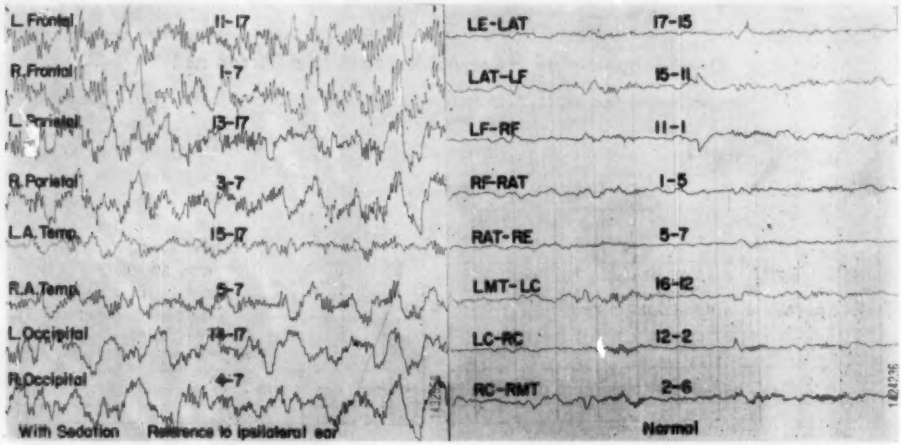
The belladonna alkaloids, scopolamine included, stimulate the brain stem mildly. Any effect on respiration is one of stimulation rather than depression.

Are Barbiturates of Benefit Before Local Anesthesia?

Sedative doses minimize mild stimulation but do not prevent convulsions. Basal narcotic doses are necessary. Barbiturates are harmful for reactions characterized by vascular collapse or respiratory failure. They do not neutralize the local anesthetic.

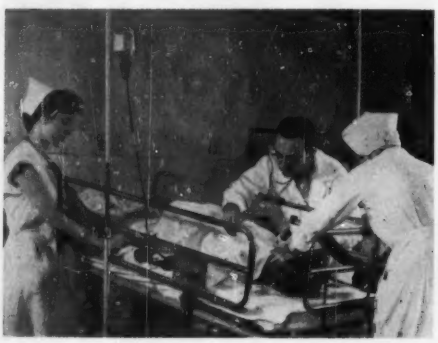
Does Premedication Reduce the Quantity of Anesthetic Needed?

Premedicants are central nervous system depressants. Therefore, they produce additive effects with anesthetics and decrease the quantity required.



Are There Disadvantages to Premedicants?

All premedicants are central nervous system depressants capable of causing hypoventilation, postural hypotension, and other untoward effects. Narcotics are more potent than hypnotics and, therefore, cause severe derangement when misused or given to excess.



What Are the Usual Errors in Premedication?

The usual errors are (1) omission of sedation the night before surgery; (2) improper timing, the drug being given too soon or too late; (3) inadequate dosage; (4) overdosage, especially in aged or poor-risk patients; (5) use of multiple agents leading to overdepression; (6) use of the wrong drug.

Experimental Analysis of Gastrojejunostomy*

HENRY N. HARKINS, M.D., Ph.D.; ROBERT V. DeVITO, M.D.; LLOYD M. NYHUS, M.D.; JOHN K. STEVENSON, M.D., and THOMAS W. JONES, M.D., Seattle

Gastrojejunostomy has failed as a treatment for active duodenal ulcer disease because of a very high relapse rate. The reasons for its failure have been clarified by experimental studies demonstrating that gastrojejunostomy increases, rather than decreases, the gastric secretion of hydrochloric acid.

These studies are best understood if the secretory responses to gastrojejunostomy are considered in terms of known mechanisms of normal gastric secretion.

I. Normal Mechanisms of Gastric Acid Secretion

Physiologist and clinicians generally accept the concept that peptic ulceration is dependent upon excess free hydrochloric acid, for only at very acid pH can peptic digestion take place. The control of acid secretion has been so extensively investigated that a comprehensive review is not possible here. A few specific experiments, however, have contributed the framework for our understanding of the physiology of gastric acid secretion.

A. The Antral Phase of Acid Secretion.

This has also been termed the chemical, hormonal, or gastric phase. In response to specific stimuli the mucosa of the gastric antrum secretes a hormone, called "gastrin," which elicits the secretion of hydrochloric acid from the parietal cells of the body (corpus) of the stomach. (It is worth emphasizing that such parietal cells are rare in the antrum itself.) Under normal conditions, this phase stimulates approximately 45% of the daily acid secretion. The following studies, among many others, are pertinent.

1. Edkins, in 1906, demonstrated that the intravenous injection of an extract of antral mucosa produced a copious flow of acid secretion from a denervated gastric pouch. He concluded that the mucosa contained "gastrin," and was the first to describe a chemical phase of gastric secretion.

2. Woodward and his co-workers, in 1950, showed that the acid secretory response of a separated, vagally denervated gastric pouch (i.e., Heidenhain pouch) to the presence of food in the stomach was markedly reduced if the antrum was excised.

3. A series of experiments done by Dr. Dragstedt's group from 1952 to 1954 further clarified the role of the antrum. These workers utilized a pouch of the antrum which was excluded from intestinal continuity, but with its innervation preserved. Mechanical distention of this pouch, or irrigation with liver extract at neutral or alkaline pH, produced prompt secretion of acid from the Heidenhain pouch. That is, these stimuli caused gastrin to be released. Alkaline pH increased the secretory response to distention, while perfusion of the antral pouch with acid abolished the response to stimulation of the antrum. These results indicated an autoregulatory mechanism of gastric acid secretion, i.e., antral stimulation causes acid secretion via the gastrin mechanism but as the pH of the antrum is lowered, gastrin release is "turned off."

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The Department of Surgery, University of Washington.

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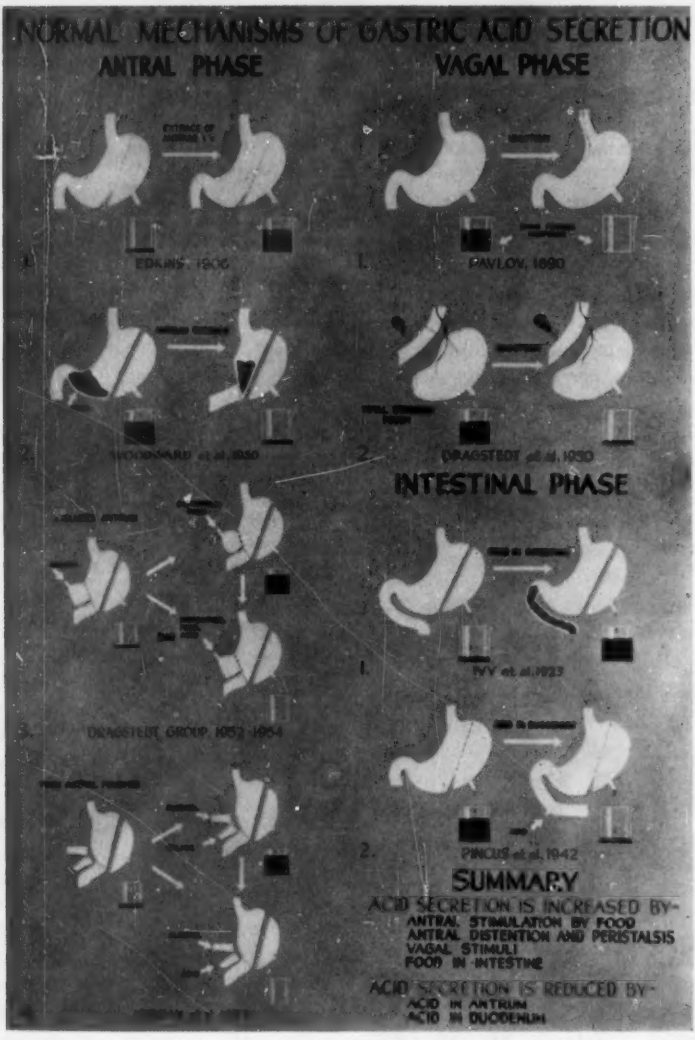
* Surgery Illustrated. Shown as a scientific exhibit at the 44th Clinical Congress of the American College of Surgeons, Chicago, Oct. 6-10, 1958.



4. The work of Jordan (1957), suggests that acid in the antrum causes the release of a substance which acts actively to inhibit parietal cell secretion of acid. His experiments were done on preparations with paired pouches made from the antrum, so that the secretory response to stimulation of the one could be reduced by perfusion of the other with acid.

B. The Vagal Phase of Acid Secretion.—The cephalic, or nervous, phase of gastric secretion is mediated by the vagus nerves. Vagal stimulation is responsible for about 45% of normal 24-hour acid secretion. This relationship has been established by a diversity of experiments, but two of the classical studies are those of Pavlov and Dragstedt.

1. Pavlov, in 1890, showed that the "appetite juice" secretory response of a dog to sham feeding was completely abolished by bilateral vagotomy.

2. Dragstedt (1950) demonstrated that the 24-hour secretion from total stomach pouches was nearly abolished by bilateral vagotomy. (Paradoxically, subsequent studies from our laboratory showed a marked increase in acid secretion from Heidenhain pouches following vagotomy of the main stomach. This is due to the increased antral stimulation, and hence potentiated hormonal phase, which occurs subsequent to the stasis produced by vagotomy.)

C. The Intestinal Phase of Gastric Acid Secretion.—Secretion of acid from denervated pouches occurs when food substances are placed in the small intestine. This mechanism, apparently humoral, was first established by Ivy in 1923, and accounts for about 10% of normal secretion.

D. The Mechanism of Duodenal Inhibition of Acid Secretion.—Pincus and his co-workers showed in 1942 that lowering the pH in the duodenum resulted in a diminution of the secretion from gastric pouches. This mechanism is apparently another means by which excess acidity is normally controlled.

In summary, secretion of acid is *increased* by stimuli carried in the vagus nerves and acting upon the parietal cells, by the release of a hormone from the antrum in response to distention or food, and by release of a humoral substance from the small intestine when stimulated by food. Secretion of acid is *reduced* when acid reaches sufficient concentration in the antrum or duodenum to lower the pH.

II. Experimental Analysis of Gastrojejunostomy

A number of experiments designed to evaluate the effect of gastrojejunostomy on acid secretion have been performed in our own laboratory. These all demonstrated that this procedure increases the secretion of acid, apparently by potentiating certain of the stimulatory mechanisms while attenuating the inhibitory mechanisms.

A. Experimental Results

1. A functioning large-stoma gastrojejunostomy increases the secretion of hydrochloric acid from Heidenhain pouches an average of 150% above control levels. Such an increase results whether the anastomosis on the stomach is high, medium, or low (to the antrum). The apparent difference between these results of our group and those of Zubiran et al., 1952, who reported that a small-stoma (<3 cm.) gastrojejunostomy produces such a stimulating effect *only when in the high or medium position,* deserves further explanation here. This clarification is especially important since the results of Zubiran et al., on the dog have repeatedly been cited as offering support for the performance of a low-stoma gastrojejunostomy in patients, so as to avoid the acid-increasing effect. In a larger group of dogs, we have performed controlled experiments indicating that, probably because of the thick rugae of the dog's antrum, a small low-stoma gastrojejunostomy of the size used by Zubiran acts just as his group said, in that it does not increase the secretion of hydrochloric acid from an associated Heidenhain pouch. At the same time, however, such a stoma does not allow barium to pass as determined by fluoroscopy, and is in essence a sham gastrojejunostomy and would not be by definition a "drainage" procedure. In this larger series of dogs we also showed that a *large* low-stoma *does* permit barium to pass and this is in essence a functioning stoma, and at the same time *does* increase the secretion of hydrochloric acid from an associated Heidenhain pouch.

We would therefore conclude, regarding this one item, on the basis of this larger series of animal experiments which are controlled both for stoma size and function as well as position, that when a gastrojejunostomy is intended to be *truly a drainage procedure,* there is no available evidence from animal experiments that it is preferable to place the stoma low on the stomach (i.e., on the antrum) because of any proved effect on gastric hydrochloric acid produc-

tion. However, for mechanical drainage purposes, we do agree that a low gastrojejunostomy may be preferable and place it thus in our practice. We merely wish to emphasize that the variations in hydrochloric acid production in the Heidenhain pouch experiments alluded to are themselves in no way a support for such a clinical procedure.

2. If antral function is reduced prior to the gastrojejunostomy by lysis of Meissner's plexus (antroneurolysis), postgastrojejunostomy hypersecretion is less marked.

3. Dogs with the antrum transplanted to the colon and with Heidenhain pouches secrete more acid from their pouches in the presence of a gastrojejunostomy as compared with a gastroduodenostomy. That is, even though the antrum is important in the production of hypersecretion by a gastrojejunostomy, other mechanisms are also involved.

4. When the occurrence of stomal ulceration with ulcerogenic stimuli is used as an index of acid production, gastrojejunostomy again is seen to increase the secretion of acid. Under the stress of either chronic histamine administration after antrectomy or of antral transplant to the colon, dogs with gastrojejunostomy develop more ulcers in a shorter period of time than do dogs with gastroduodenostomy.

5. Following vagotomy to the main stomach, Heidenhain pouch hypersecretion occurs because of gastric stasis. When a gastrojejunostomy is added as the drainage procedure, hypersecretion persists; Finney pyloroplasty returns secretion toward control levels.

B. Physiologic Analysis of Gastrojejunostomy.—Gastrojejunostomy was first used by Wölfler in 1881 as a drainage procedure for pyloric carcinoma, and by Rydygier in 1884 for obstructing duodenal ulcer. It rapidly came into favor as *the* procedure for duodenal ulcer disease after the turn of the century, and was done with three purposes:

1. To shunt acid from the area of ulceration
2. To neutralize acid in the stomach
3. To drain an obstructed stomach

The great majority of the original duodenal ulcers do heal after gastrojejunostomy, but the incidence of subsequent jejunal stomal ulcers is very high. In 1925, Lewisohn reported the first large series followed for a number of years, and demonstrated that 34% of patients with gastrojejunostomy ultimately developed stomal ulcers. A more contemporary report (Clark, 1951) records a relapse rate of 37.5%. Why does gastrojejunostomy fail?

When a functioning gastrojejunostomy is formed, the passage of substances in the region of the stoma is altered in several ways.

1. A portion of the food mass is shunted directly into the efferent loop, while a remnant continues to be carried into the duodenum by peristalsis.

2. Secreted gastric acid, being fluid, tends to pass directly into the efferent loop.

3. Alkaline duodenal juices pass through the afferent loop and into the distal stomach.

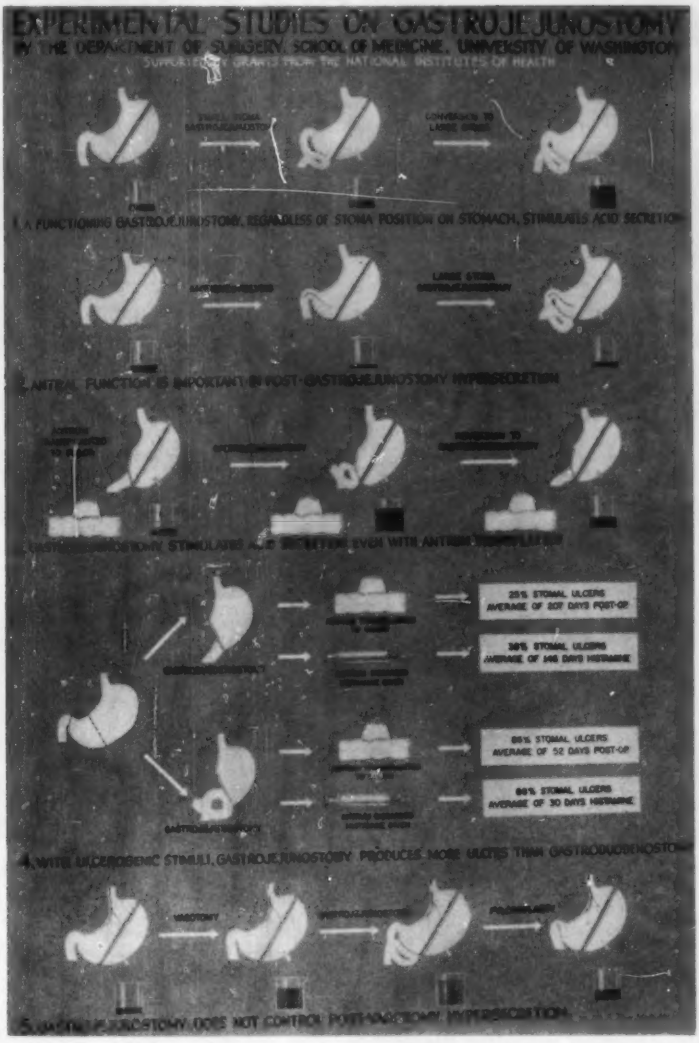
4. Food entering the duodenum is returned to the stomach via the afferent loop; peristalsis again carries a portion into the duodenum; i.e., a circus movement of food material is established.

The resultant modifications of normal mechanisms of acid secretion are as follows:

1. The passage of food directly into the small intestine through the gastrojejunostomy augments the intestinal phase of gastric secretion, and acid production is *increased.*

2. The shunting of acid from the antrum and duodenum reduces the normal autoregulatory and inhibitory functions served by acid, so that HCl secretion tends to proceed unchecked, and acid production is *increased.*

3. The reflux of alkaline fluid into the stomach neutralizes any acid reaching the antrum so that inhibitory mechanisms are further reduced, and the resultant alkaline pH of the antrum acts to potentiate antral


EXPERIMENTAL STUDIES ON GASTROJEJUNOSTOMY
BY THE DEPARTMENT OF SURGERY, SCHOOL OF MEDICINE, UNIVERSITY OF WASHINGTON
SUPPORTED BY GRANTS FROM THE NATIONAL INSTITUTES OF HEALTH
1. A FUNCTIONING GASTROJEJUNOSTOMY, REGARDLESS OF STOMA POSITION ON STOMACH, STIMULATES ACID SECRETION
2. ANTRAL FUNCTION IS IMPORTANT IN POST-GASTROJEJUNOSTOMY HYPERSECRETION
25% STOMAL ULCERS
AVERAGE OF 207 DAYS POST-OP
36% STOMAL ULCERS
AVERAGE OF 148 DAYS HISTAMINE
85% STOMAL ULCERS
AVERAGE OF 52 DAYS POST-OP
80% STOMAL ULCERS
AVERAGE OF 30 DAYS HISTAMINE
4. WITH ULCEROGENIC STIMULI, GASTROJEJUNOSTOMY PRODUCES MORE ULCERS THAN GASTRODUODENOSTO
5. GASTROJEJUNOSTOMY DOES NOT CONTROL POST-VAGOTOMY HYPERSECRETION

stimulation by other mechanisms. Hence, acid production is *increased*.

4. Circus movement of food results in repetitive stimulation of the antrum by the same mass of food, so that acid production is *increased*.

5. The vagal phase of acid secretion is not altered.

Summary

It is apparent from the above that gastrojejunostomy acts to *increase* acid production through all mechanisms other than vagal, with a resultant high incidence of jejunal stomal ulceration. While antral mechanisms are prominent in the production of post-gastrojejunostomy hypersecretion, stimulation of secretion occurs through alteration of the duodenal and intestinal phases even in the absence of the antrum.

Conclusions

Gastrojejunostomy remains valuable as an expedient drainage procedure for the quies-

cent but obstructing ulcer; we submit, however, that it has little place in the surgical therapy of the active duodenal ulcer.

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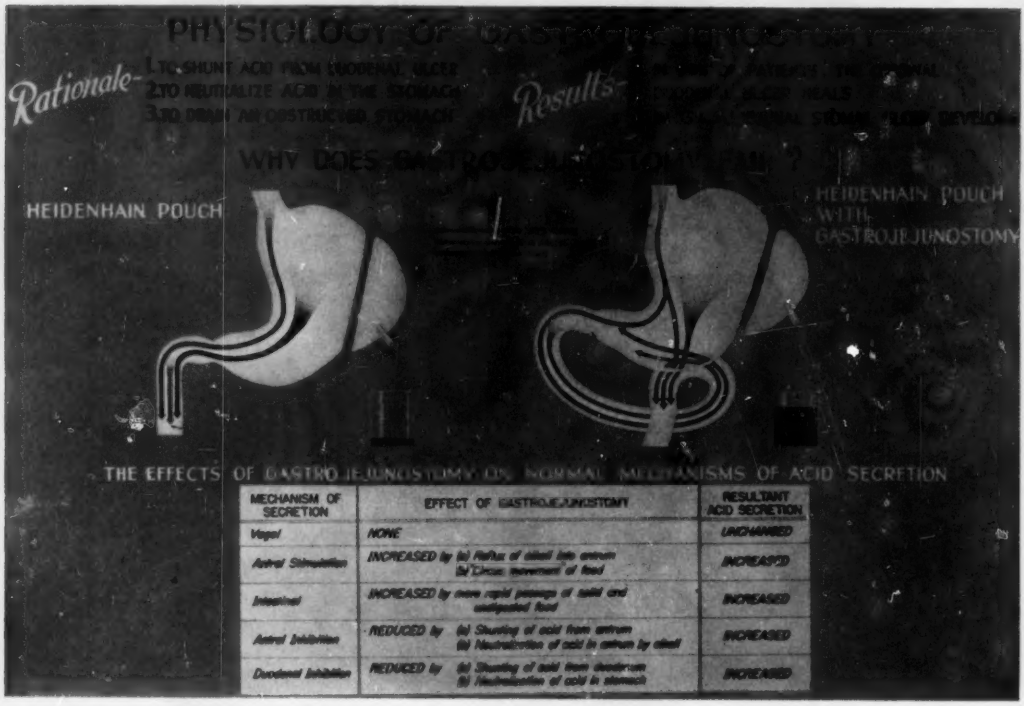

Hirschsprung's Disease in the Newborn*

ORVAR SWENSON, M.D., and JOHN H. FISHER, M.D., Boston

Hirschsprung's disease is one of the more frequent causes of intestinal obstruction in the newborn. Improvements in the methods of diagnosis and management of patients in this group have increased the survival rate. The 3-day-old infant in Figure 1 has the signs of acute intestinal obstruction. He has become dehydrated and lethargic from fluid and electrolyte loss due to repeated bile-stained vomiting. In addition there is abdominal distension and obstipation. Many of the infants with Hirschsprung's disease present with symptoms and signs indistinguishable from other cause of intestinal obstruction such as ileal atresia. Table 1 compares the incidence of vomiting, obstipation, and abdominal distension in a group of neonates with Hirschsprung's disease and another group with ileal atresia. Figures 2 through 5 indicate the proper diagnostic methods to differentiate Hirschsprung's disease from other causes of acute intestinal obstruction in the newborn. Prompt abdominal exploration is needed in small bowel obstruction, but an abdominal exploration imposes an unnecessary risk on the infant with Hirschsprung's disease. Figures 6 through 8 suggest the need of an additional diagnostic test in those infants suspected of having Hirschsprung's disease. There are certain conditions, such as meconium plug syndrome or cretinism, that have the symptoms of intestinal obstruction, and may have roentgenographic findings suggestive of Hirschsprung's disease. Those patients having roentgenographic findings suggestive of Hirschsprung's disease should have a rectal biopsy performed to rule out these other conditions. Rectal biopsy is also indicated in certain infants who have the symptoms of Hirschsprung's disease and yet do not have characteristic roentgenographic findings. Table 2 indicates that the roentgenographic study does not completely rule out the diagnosis. Figures 9 through 14 present the operative technique to obtain the rectal biopsy. The photomicrographs in Figures 15 and 16 indicate the histologic criteria for the diagnosis of Hirschsprung's disease in the newborn. Table 3 outlines the steps in the management of the newborn with Hirschsprung's disease. In most of the patients the aganglionic lesion extends

TABLE 1.—*Incidence of Symptoms in Hirschsprung's Disease and in Ileal Atresia*

	Congenital Megacolon			Ileal Atresia		
Symptom	Total	Cases with Symptom	% with Symptoms	Total	Cases with Symptom	% with Symptom
Vomiting	55	27	50	10	9	90
Obstipation	55	40	75	10	8	80
Abdominal distension	55	38	70	10	10	100

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From The Boston Floating Hospital for Infants and Children, and Tufts University School of Medicine. Professor of Pediatric Surgery (Dr. Swenson); Assistant Professor of Surgery (Dr. Fisher), Tufts University School of Medicine.

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TABLE 2.—*Cases Diagnosed by Roentgenography*

	Total	Cases Diagnosed	% of Cases Diagnosed
Plain roentgenogram	42	6	15
Barium enema roentgenogram	58	48	84
Rectal biopsy	10 *	10	100

* In these cases, barium enema failed to establish a diagnosis.

no higher than the sigmoid colon. In these cases a small rectal tube may be passed beyond the aganglionic segment and the intestinal obstruction relieved by colonic irrigations using an Asepto syringe and small quantities of saline solution. An elective colostomy in the descending colon can then be done when the child is several days old, after the diagnosis of Hirschsprung's disease has been substantiated by rectal biopsy. Infants with Hirschsprung's disease are very susceptible to diarrheal disease. They are prone to episodes of vomiting and abdominal distension with or without diarrhea. They may rapidly suffer severe fluid and electrolyte loss through fluid collecting in the dilated colon. When an infant is referred with this problem, it is best to manage the diarrheal disease conservatively with intravenous fluids, in addition to colonic irrigations as described above to empty the colon of the liquid stool. A colostomy may be done after the diagnosis is established by rectal biopsy.

The definitive resection of the aganglionic segment and the colostomy are carried out when the child is 25 to 30 lb. in weight.

TABLE 3.—*Management*

1. Relieve acute intestinal obstruction with rectal irrigations.
2. Treat enteritis with rectal irrigations, nothing by mouth, and intravenous feedings.
3. Colostomy, temporary, elective, as soon as the diagnosis is made by rectal biopsy.
4. Resection of colostomy and aganglionic segment with pull-through anastomosis when child is 25 to 30 lb. in weight.

Table 4 lists the mortality risks with various methods of management. The preferred method of management is an elective colostomy after rectal biopsy has established the diagnosis. The technique for colostomy is presented in Figures 17 through 21. The colostomy is placed near the distal end of the

TABLE 4.—*Results*

	% Mortality
1. Nonoperative management	70
2. Early resection	15
3. Colostomy during enteritis	25
4. Elective colostomy	4
5. Resection of aganglionic segment after 25 lb.	0

aganglionic segment so that it may be resected with the aganglionic segment when the child is 25 to 30 lb. in weight. A biopsy of the colon wall is secured at the time of colostomy to be certain that ganglion cells are present in this segment. If the colostomy is placed in an aganglionic segment, it will not function properly.

20 Ash St., (11).

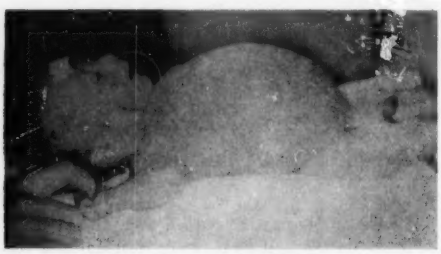


Fig. 1.—Photograph of a newborn infant with Hirschsprung's disease. Note the signs of acute intestinal obstruction.

Fig. 2.—Plain roentgenogram indicating intestinal obstruction. It is impossible to determine whether the dilated loops are small or large intestine.

Fig. 3.—Barium enema in the same patient identifies dilated loops to be colon. Small-bowel obstruction is thus excluded. A diagnosis of Hirschsprung's disease is suggested but can be established only by rectal biopsy.

Fig. 4.—Plain roentgenogram of an infant with intestinal obstruction. Again it is impossible to decide whether this is Hirschsprung's disease or small-bowel obstruction.

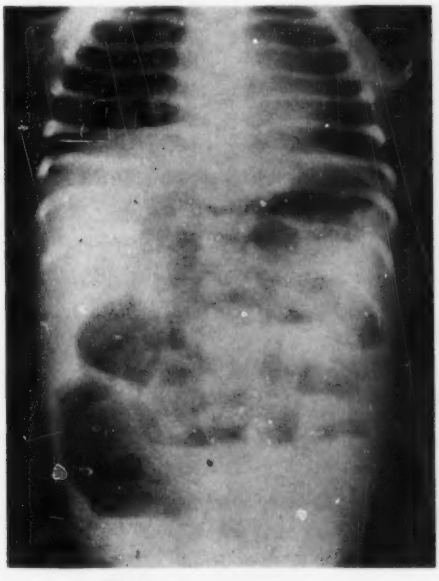


Fig. 5.—Barium enema in the same patient reveals a "microcolon." Dilated intestine is small bowel. Thus the diagnosis of mechanical obstruction is established. This patient needs prompt abdominal exploration.

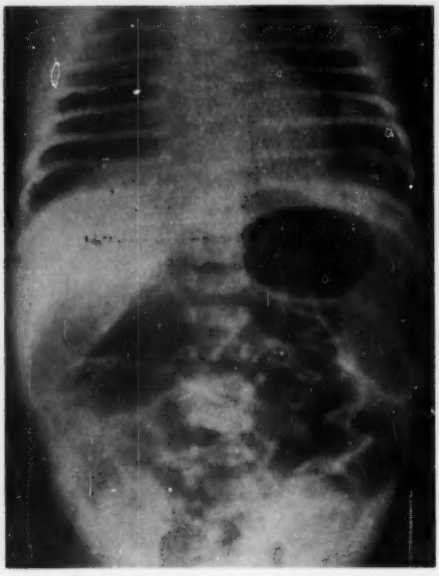


Fig. 6.—Roentgenogram of a patient with the symptoms of intestinal obstruction.

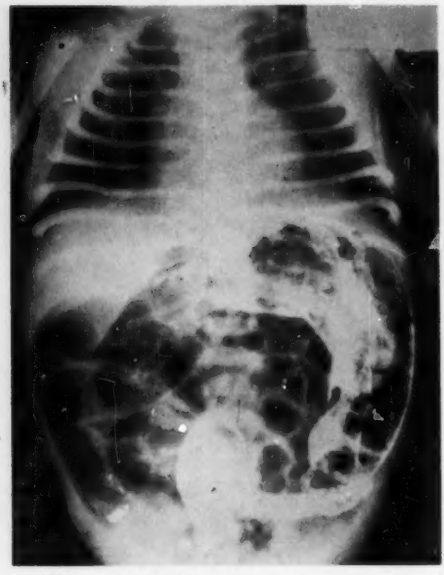


Fig. 7.—The barium enema is suggestive of Hirschsprung's disease. The rectal biopsy contained ganglion cells, thus excluding this diagnosis. This patient proved to have a meconium plug.

Fig. 8.—Barium enema in a newborn suggesting the diagnosis of Hirschsprung's disease. Note the narrow distal segment. Rectal biopsy specimen contained ganglion cells. This baby proved to be a cretin.

Fig. 9.—The patient is placed on his side; rectal dilatation is accomplished, and a right-angle retracter is inserted. (Reproduced from Swenson O.: Pediatric Surgery, 1958, courtsey of Appleton-Century-Crofts, Inc., New York.)

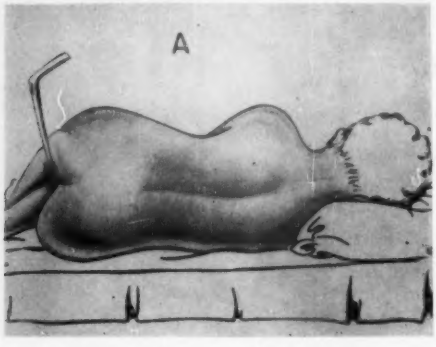
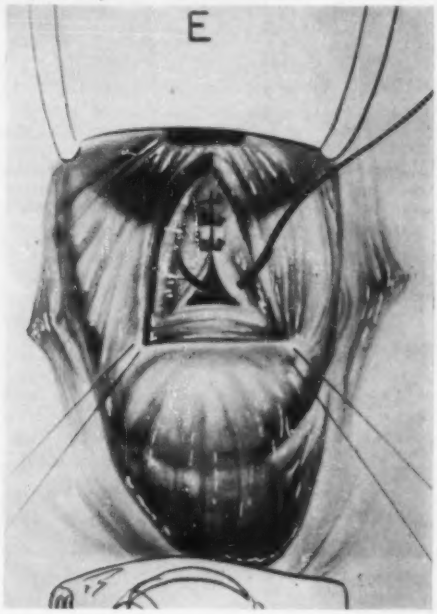


Fig. 10.—The rectal wall is pulled downward by digital pressure on a sponge and with two traction sutures of silk. An incision through the mucosa is made beginning 3 cm. above the mucocutaneous margin. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

Fig. 11.—The mucosa has been separated from the muscular layers by blunt dissection. The muscular layers are separated from the perirectal tissue. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

Fig. 12.—A triangular area of the muscular wall is removed, measuring at least 1 cm. in length. This must include the circular and longitudinal layers. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

Fig. 13.—The defect in the muscular wall is closed with interrupted 00000 silk sutures. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)



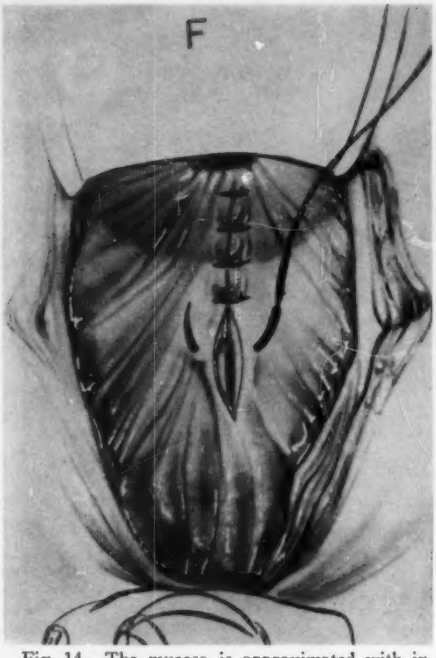


Fig. 14.—The mucosa is approximated with interrupted chromic catgut sutures. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

Fig. 15.—Photomicrograph of a rectal biopsy section. Ganglion cells in Auerbach's plexus exclude the diagnosis of Hirschsprung's disease.

Fig. 16.—Photomicrograph of a rectal biopsy section containing no ganglion cells. This establishes the diagnosis of Hirschsprung's disease.

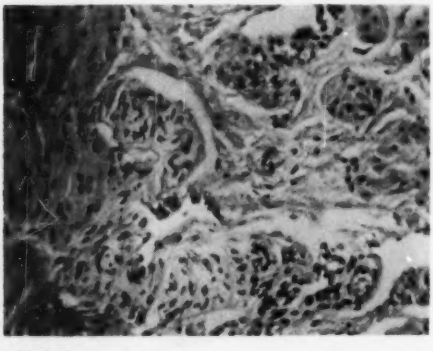


Fig. 17.—Diagram indicating position of rectus muscle-splitting incision. When the lesion extends into the transverse colon, the incision is placed in the appropriate quadrant of the upper abdomen. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

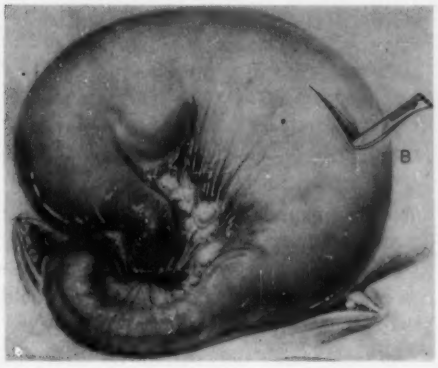


Fig. 18.—Dilated sigmoid is delivered and an incision made through the muscular coats about 10 cm. from the funnel area. Care is taken not to open the mucosa. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

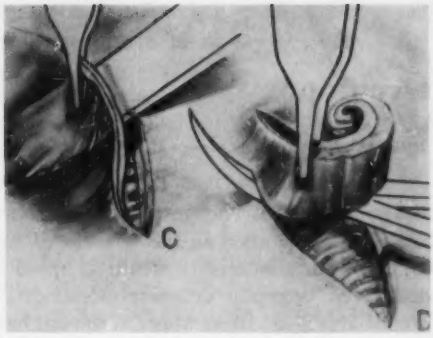


Fig. 19.—Muscular coats are separated from the mucosa by blunt dissection on one side of the incision. The edge of the muscular wall is excised. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

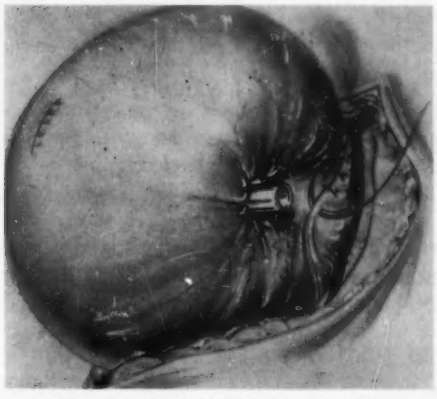


Fig. 20.—Edges of incised muscular layer are approximated with interrupted silk sutures. A rod is placed through the mesentery adjacent to the bowel wall. Suturing of the peritoneum to the colon is necessary to prevent dehiscence of small intestine beside the colostomy. (Reproduced from Swenson O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

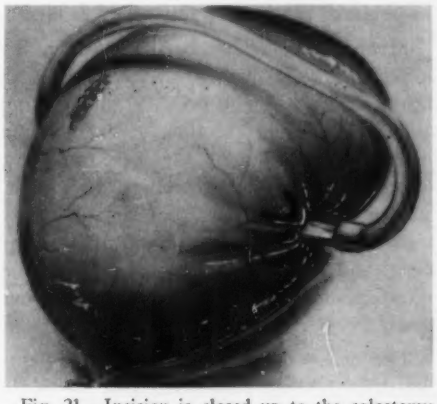


Fig. 21.—Incision is closed up to the colostomy with interrupted silk sutures and rod secured by attaching a rubber tube. Colostomy is opened 12 to 24 hours after the colostomy is made. (Reproduced from Swenson, O.: Pediatric Surgery, 1958, courtesy of Appleton-Century-Crofts, Inc., New York.)

Orchiopexy*

JOHN L. KEELEY, M.D., Chicago

The absence of one or both testes from the expected location in the scrotum of an infant is an abnormality which is usually discovered promptly by most mothers. It occurs in 1 in 10 newborn infants, but spontaneous descent during the first year of life reduces this incidence to about 1 in 50. These figures suggest that orchiopexy during this period is not indicated. A decreased rate of spontaneous descent obtains from the age of one year until adolescence, and in this interval the question of whether to perform orchiopexy must be considered.

Much emphasis has been placed on the importance of distinguishing between a testis which has not been in the scrotum and one which at the time of examination is merely retracted by vigorous cremaster fibers in response to chilling or fright. Information obtained from an observant mother, repeated examination of the patient, and the state of development of the scrotum should settle this question without much trouble.

The cause of undescended testis is undetermined. One view holds that the testis does not reach a normal position because it is not a normal gland to begin with, as indicated by the lack of gross development or the presence of degenerative changes in its microscopic appearance. An opposing view suggests that the changes are due to unfavorable higher environmental temperatures to which the potentially normal abdominal testis is subjected. There is a general agreement that gonadotrophic hormone is a significant factor in the normal descent of the testes. However, there is a growing belief that the testes which descend after hormonal treatment would have reached a normal position anyway.

Surgeons have not shared the view of others regarding the effectiveness of hormonal therapy in causing testicular descent. When one testis descends and the other does not, factors other than hormonal must be considered. The attachments which must be freed during orchiopexy indicate that only by surgical intervention could the testis be brought into the scrotum in this group of patients.

It is agreed that the most important function of the testes is spermatogenesis, which may be evaluated by testicular biopsy or by eventual proof of fertility. On the basis of testicular biopsy it has been shown that the unilateral undescended testis develops at the same rate as its fellow up to the age of 8 to 10 years. Spermatogenesis in the undescended testis is difficult to assess on the basis of fertility in patients with a grossly normal scrotal testis on the other side. However, fertility in patients with bilateral undescended testes treated by orchiopexy is considered well established and indicates that effective spermatogenesis is a definite possibility after orchiopexy.

There is no question at present concerning the influence of temperature on spermatogenesis. Transplantation of a functioning testis into the abdomen of experimental animals stops spermatogenesis. It may also be interrupted in humans after a febrile illness. The application of heat to the scrotum has had the same effect. During hibernation in animals, the testes are withdrawn into the abdomen and then show degeneration. Regeneration occurs when

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From the Departments of Surgery, Stritch School of Medicine of Loyola University, Mercy Hospital, and Cook County Hospital.

* Surgery Illustrated.

the testes return to the scrotum during the mating season.

It has been shown by Dahl and Herrick that, in addition to the lower environmental temperature of the scrotum, testicular temperature is influenced by the counterexchange of heat between the internal spermatic artery and the pampiniform plexus. The tortuosity of both the pampiniform plexus and the spermatic artery contributes to the efficiency of this counterexchange of heat. Any alteration in this anatomical arrangement, either by excision of the pampiniform plexus or by disturbing the tortuosity of the spermatic artery, should therefore be avoided. In addition to providing suitable environmental temperature, other indications for orchiopexy must be considered. Those most often mentioned are correction of an associated patent tunica vaginalis or a fully developed hernia, decreasing the risk of physical or psychic trauma, and preventing possible malignant degeneration of the testis, although this potentiality is debatable.

The future of an undescended testis is not at present sufficiently predictable for most surgeons to refuse operation when confronted by anxious parents who want something done to make their son "normal."

The operation should be one which preserves the blood supply of the testis and the vascular arrangement described above for arteriovenous counterexchange of heat. Therefore the necessary dissection should involve the retroperitoneal structures rather than those close to the testis. Simultaneous bilateral orchiopexy should not be done. Eunuchism will result if atrophy of both testes occur. Successful orchiopexy should be accomplished on one side before the second side is operated upon. Unilateral cryptorchism is five times as frequent as bilateral failure of descent. The optimum age for operations is in the period between 4 and 8 years.

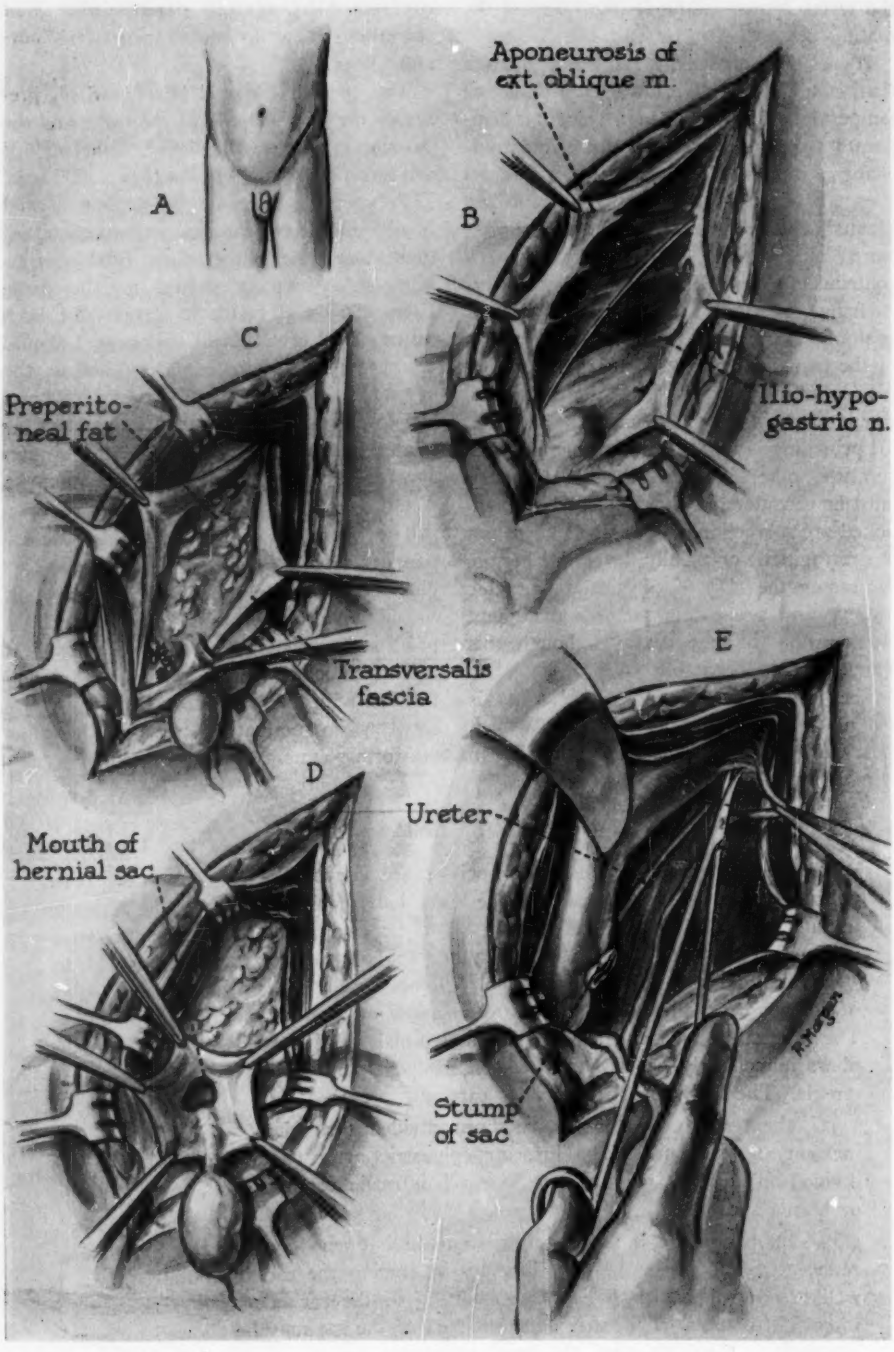
The procedure presented in the accompanying illustrations is designed to accomplish three objectives: (1) the preservation of effective blood supply; (2) a scrotal position for the testes without tension on its attachments, and (3) minimal interference with the counterexchange of heat between the spermatic artery and the pampiniform plexus.

A, the location and extent of the incision are shown. When no testis has been palpated in the inguinal canal only the lower half of the incision should be made initially. Thus, if no testis is present, or only a small nubbin of testicular tissue is found, the longer incision necessary for the complete operation is not made.

B, the aponeurosis of the external oblique muscle is divided in the direction of its fibers distally into the external inguinal and proximally into the muscle. The internal oblique and transversus abdominis muscles are divided across their fibers immediately beneath the incision in the external oblique aponeurosis and muscle. The iliohypogastric nerve is carefully preserved.

C, the transversalis fascia has been divided throughout the length of the incision and retracted. The inferior epigastric artery and vein(s) have been divided and ligated individually. A search is made for a patent tunica vaginalis or hernia sac.

D, the hernia sac or patent tunica vaginalis is opened and the contents, if any, reduced. Should there be any difficulty in freeing the sac from the cord, it is well to remember that the proximal portion of the sac is usually less adherent to cord structures. The more adherent part of the sac may be allowed to remain attached to the cord until the cord structures are further mobilized and the


A
B
Aponeurosis of ext. oblique m.
Ilio-hypo-gastric n.
C
Preperito-neal fat
Transversalis fascia
D
Mouth of hernial sac
E
Ureter
Stump of sac

necessity for further separation of the sac from the cord to provide adequate length is demonstrated.

The hernia sac is closed. The empty hernia sac may be twisted and a silk suture passed through the twisted portion two or three times and tied. Tears in a friable sac wall can be avoided in this way.

E, adequate exposure is the key to successful orchiopexy. It is obtained by retracting the peritoneum medially and anteriorly. All fibers adherent to the spermatic vessels must be divided with fine, sharp scissors under direct vision. The division of loose areolar tissue in the retroperitoneal space mobilizes the spermatic vessels with less danger of hematoma than dissection of these structures near the testicle.

F, the dissection is carried to the level of the lower pole of the kidney to free the spermatic vessels. It is sometimes necessary to free the vas deferens in a similar manner, although it is usually the vessels which hold the testicle at a high level. An anchoring stitch of No. 0 silk has been placed through the lower pole of the testicle. The free ends of the silk suture will pass through the scrotal wall at one point.

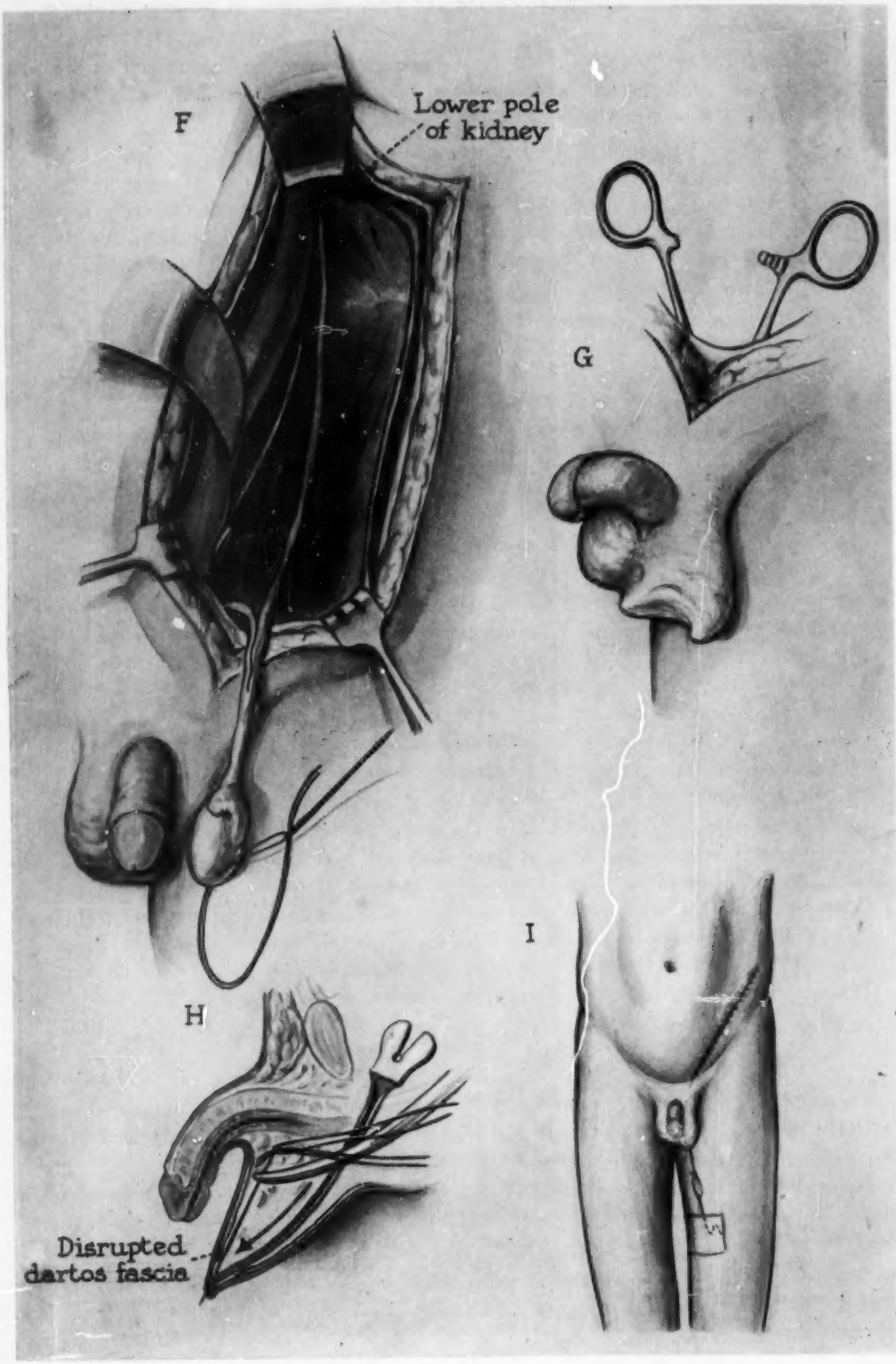
G, since the corresponding side of the scrotum has not contained the testicle before, it is usually small. A space is made for the testicle by divulsing Dartos fascia and stretching the scrotal skin in the manner shown.

H, guided by the finger, a grooved director is passed along the path made to the most dependent portion of the scrotum. The grooved director is bent in a gentle curve so that the tip of the needle in the groove can be guided to the end of the director. The tip of the grooved director is bent so that the needle will slide out of the groove and immediately penetrate the scrotal wall at the selected point. It must be emphasized that the purpose of this traction suture is *not* to put tension on the vas deferens or spermatic vessels. It is simply to maintain the position of the testis in the most dependent portion of the scrotum to which it will become adherent in a few days.

I, accurate approximation of all layers of the abdominal wall is done. Starting with the transversalis fascia, each layer is closed by interrupted sutures of fine silk. A rubber band is interposed between the silk suture in the lower pole of the testicle and another silk suture fastened to the anteromedial surface of the thigh. This arrangement is applied in such a manner that there is only slight tension on the rubber band. By the time the patient is ready to leave the hospital (after five to seven days), the traction suture may be removed.

When this technique is carefully executed the testicle can be placed in the scrotum without significant tension on the vas deferens and the testicular blood supply in all but a few patients. In these, anchoring the testis as low as possible as the first part of a two-stage procedure may be done. A second operation to place the testes in the scrotum is occasionally successful. Orchiectomy is recommended by some authors if the testis cannot be placed in the scrotum.

706 S. Wolcott Ave. (12).


F
Lower pole
of kidney
G
H
Disrupted
dartos fascia
I

Postbulbar Duodenal Obstruction*

CHARLES H. BROWN, M.D., and WILLIAM C. STRITTMATTER, M.D., Cleveland

CONGENITAL LESIONS

Compression of duodenum by superior mesenteric artery; duodenal ileus.

Superior mesenteric artery compression of duodenum.

Superior mesenteric artery compression of duodenum.

Malrotation of small intestine with volvulus.

Congenital band at ligament of Treitz.

Annular pancreas.

Congenital band at third portion of duodenum.

Annular pancreas.

Submitted for publication Aug. 25, 1959.

From the Department of Gastroenterology and the Department of Radiology, The Cleveland Clinic Foundation and The Frank E. Bunts Educational Institute.

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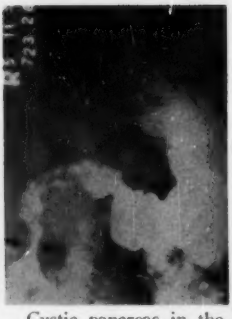
EXTRINSIC LESIONS

Aneurysm of aorta compressing duodenum (third part).

Duodenal loop compressed by pancreatic pseudocyst.

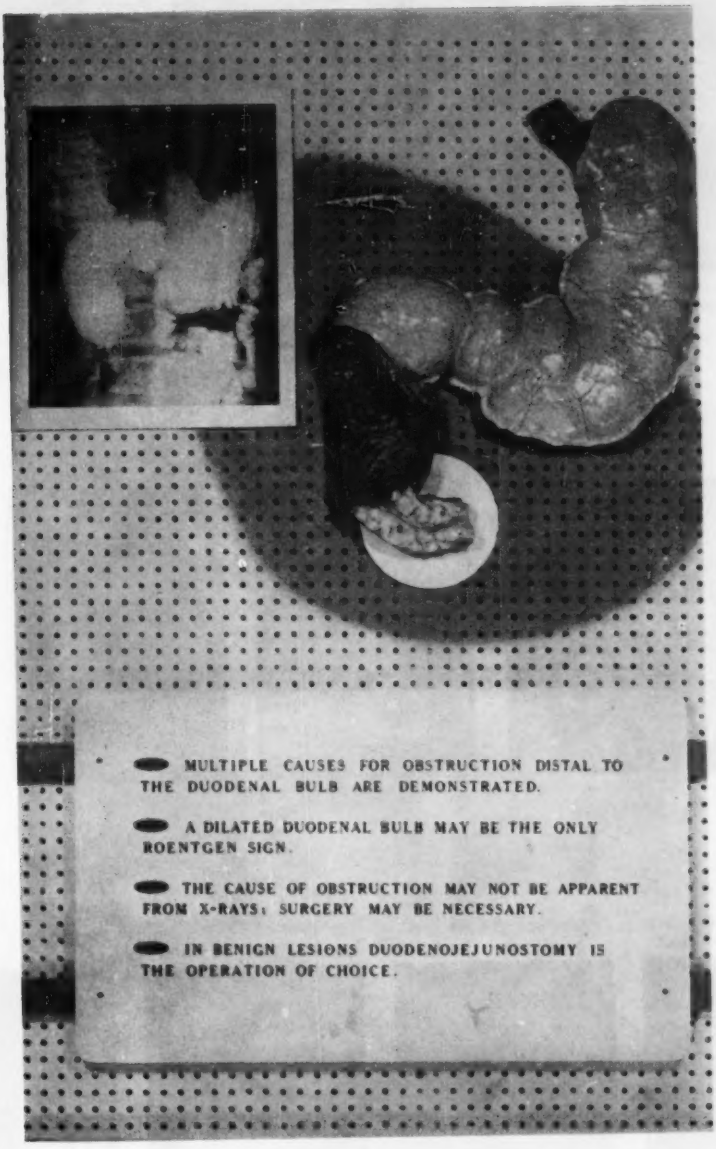


Cystic pancreas in the wall of the duodenum.

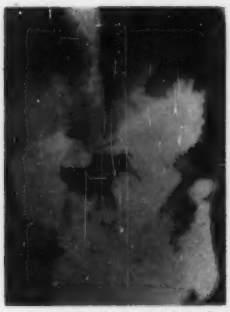
Islet-cell adenoma of head of pancreas; dilated duodenal bulb.

Carcinoma of the pancreas causing obstruction at ligament of Treitz.

Carcinoma of pancreas.


MULTIPLE CAUSES FOR OBSTRUCTION DISTAL TO THE DUODENAL BULB ARE DEMONSTRATED.
A DILATED DUODENAL BULB MAY BE THE ONLY ROENTGEN SIGN.
THE CAUSE OF OBSTRUCTION MAY NOT BE APPARENT FROM X-RAYS; SURGERY MAY BE NECESSARY.
IN BENIGN LESIONS DUODENOJEJUNOSTOMY IS THE OPERATION OF CHOICE.

INTRINSIC LESIONS



Stomal ulcer with adhesions obstructing the afferent loop (duodenum).

Primary jejunal ulcer.

Carcinoma of duodenum.

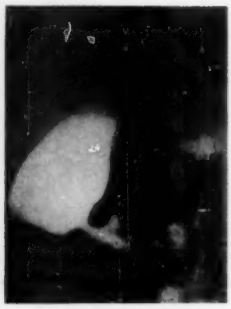
Carcinoma of duodenum.

Postbulbar ulcer.

Postbulbar ulcer.

Duodenitis (regional enteritis).

Duodenitis (regional enteritis).



INTRINSIC LESIONS

Carcinoma of duodenum.

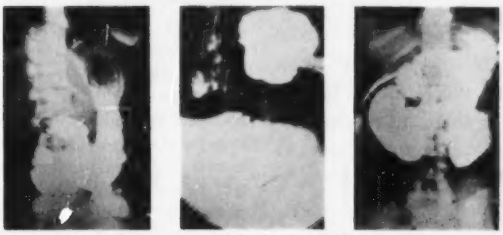
Carcinoma of duodenum.

Reticulum-cell sarcoma.

Lymphosarcoma.

Hodgkin's sarcoma.

MISCELLANEOUS

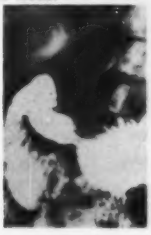


Lupus erythematosus with duodenal ileus, and one year later—perforated ulcer.

Hematoma at ligament of Treitz.

Acute angle at ligament of Treitz.

Lupus erythematosus with duodenal ileus.



The Effect of Pancreatic Fistula on Gastric Secretion

HERBERT B. GREENLEE, M.D.; THOMAS S. NELSEN, M.D., and LESTER R. DRAGSTEDT, M.D., Ph.D., Chicago

Zollinger and Ellison first called attention to instances of recurrent peptic ulceration associated with islet cell tumors of the pancreas.[1] These were characterized by hyperacidity and hypersecretion of acid gastric juice and not controlled by vagotomy and extensive gastric resection. Previous experimental studies have also demonstrated an interrelationship between the pancreas and gastric secretion. Dragstedt reported in 1942 that approximately 100% of dogs with external total pancreatic fistulae developed duodenal ulcer, whereas dogs in whom the pancreatic ducts had been ligated had only a 29% incidence of duodenal ulcer. He further found that dogs upon whom a pancreatectomy had been performed developed ulcer in only 1%-3%.[2] In all three of these preparations the external secretion of the pancreas is absent from the duodenum.

The internal drainage of bile and pancreatic juice to the terminal ileum in the Mann-Williamson dog also results in a high incidence of peptic ulceration. Recent studies would indicate that this high incidence of ulceration is not entirely due to lack of neutralization of acid gastric juice by pancreatic juice and bile, but also to an increased output of acid gastric juice.[3]

The purpose of the present experiments was to determine quantitatively the effect of total external pancreatic fistula on the output of acid gastric juice in previously controlled Heidenhain pouch dogs.

Submitted for publication June 12, 1959.

From the Department of Surgery of The University of Chicago.

This work has been aided by grants from the Otho S. A. Sprague Memorial Institute and from the Division of Research Grants and Fellowships, National Institutes of Health.

Method

Denervated pouches of the fundus of the stomach (Heidenhain pouch) were constructed in five healthy mongrel dogs. After recovery from the operative procedure, quantitative 24-hour collections of gastric secretion from the pouches were obtained and the daily output of free HCl determined by titration of the gastric juice against 0.1 N solution of sodium hydroxide, using Topfer's reagent as the indicator. Results are expressed in milliequivalents of free hydrochloric acid per unit time (volume in liters $\times$ concentration in clinical units = mEq. of acid). After the control period, varying from three to five weeks, the dogs were reoperated on and an external total pancreatic fistula constructed in the following manner. The large lower pancreatic duct was exposed and identified. The remainder of the pancreas was then dissected free from the duodenum in order to interrupt any accessory communications between pancreas and duodenum. The duodenum was then transected 1.5-2 cm. proximal and distal to this main pancreatic duct. Both ends of this duodenal segment were closed and a stainless steel cannula inserted into this pouch. The cannula was then exteriorized via a separate stab wound. Duodenal continuity was reestablished by an end-to-end anastomosis of the transected duodenum (Fig. 1).

The collections were discarded until one week postoperatively when the dogs were eating a standard laboratory diet. It was found necessary to replace electrolytes by parenteral administration in order to keep the animals alive and in electrolyte balance. When this experiment was begun, there was 100% mortality due to inadequate electrolyte replacement. The animals died within one week from severe acidosis and dehydration. It was found that in order to maintain an animal in good electrolyte balance, it was necessary to administer intravenously approximately 500 cc. of Ringer's solution and 400 cc. of ⅙ M sodium lactate daily. On this regimen an animal could be maintained in good electrolyte balance by varying the total amount of parenteral replacement slightly, depending on the pancreatic juice and Heidenhain pouch output.

Results

Following construction of the pancreatic fistula, the gastric secretory output from

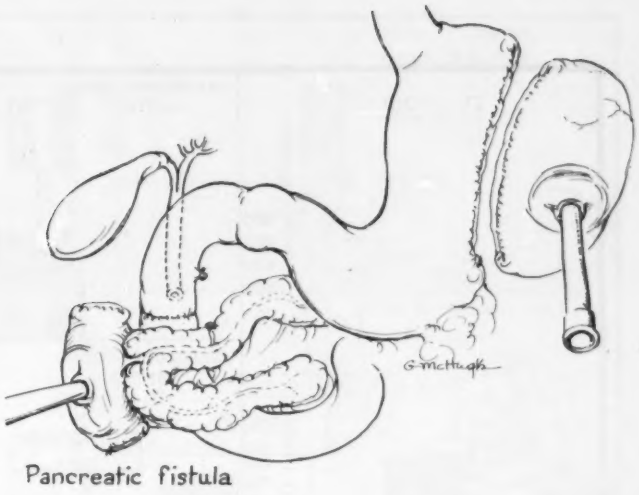


Fig. 1.—Artist's depiction of the pancreatic fistula-Heidenhain pouch preparation.

the Heidenhain pouch increased in all dogs (Fig. 2), showing over a 400% increase in two of the dogs (Figs. 3 and 4). The volume of pancreatic juice secreted by any one dog remained relatively constant. There was no direct correlation between pancreatic juice output and the increase in Heidenhain pouch output. This preparation responds to intravenous injection of pancreatic secretion (1 unit kg.)[4] with a rise in output of pancreatic juice (Fig. 5).

Comment

The explanation for the rise in the Heidenhain pouch output following construction of the pancreatic fistula could be due to one of at least three possibilities—(1) electrolyte disturbance, (2) loss of pancreatic juice from the duodenum, (3) hormonal effect from the pancreas.

The possibility has been considered that the marked rise in Heidenhain pouch output

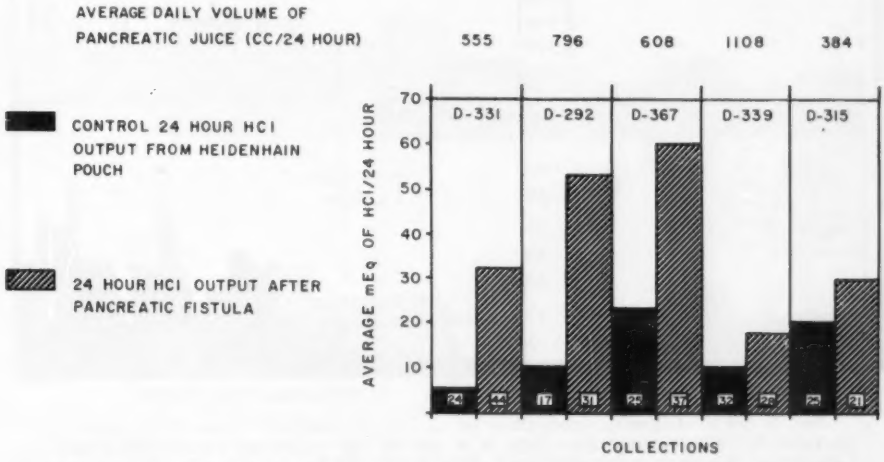
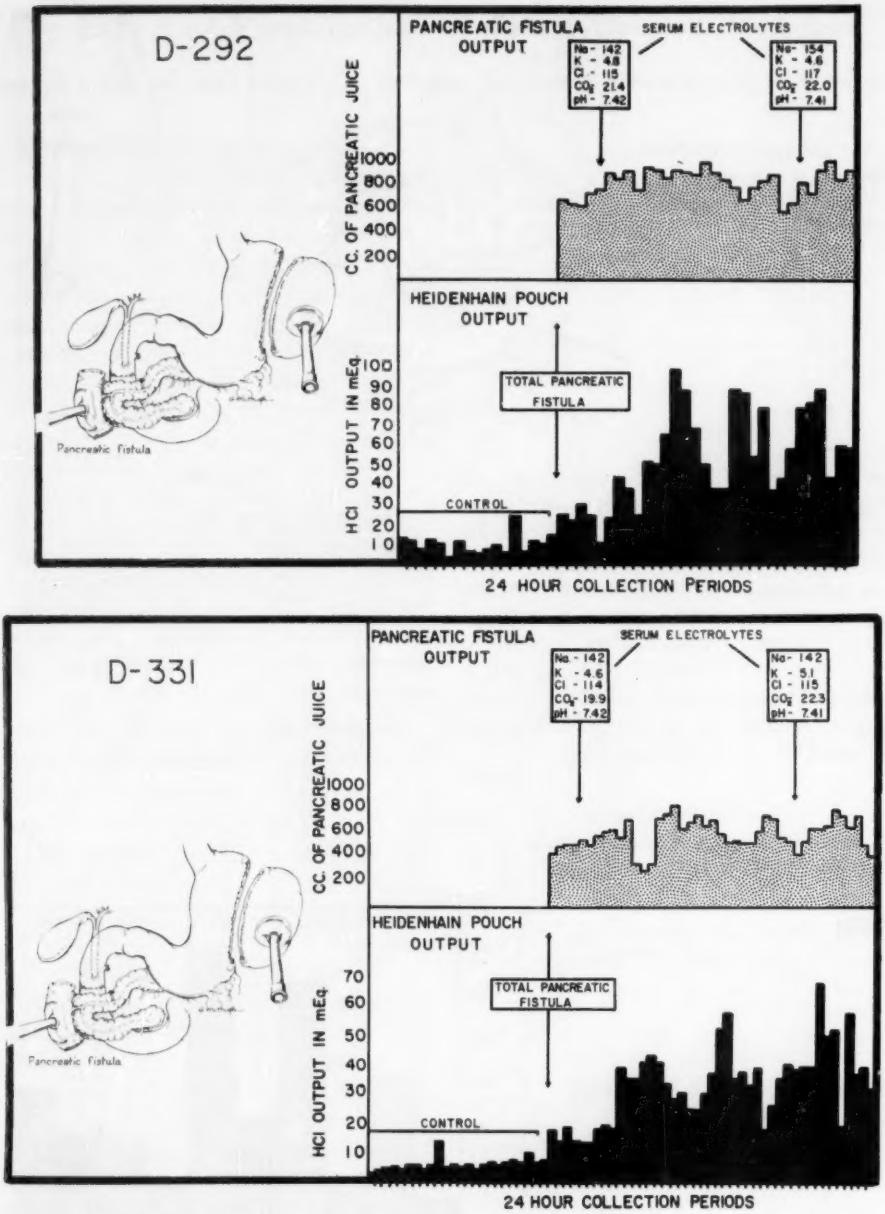


Fig. 2.—Average daily gastric secretory output in five dogs after construction of total pancreatic fistula.



Figs. 3 and 4.—Following construction of an external pancreatic fistula in a previously controlled Heidenhain pouch dog, there is a marked rise in the gastric secretory output. Examples of this increase are graphically demonstrated above.

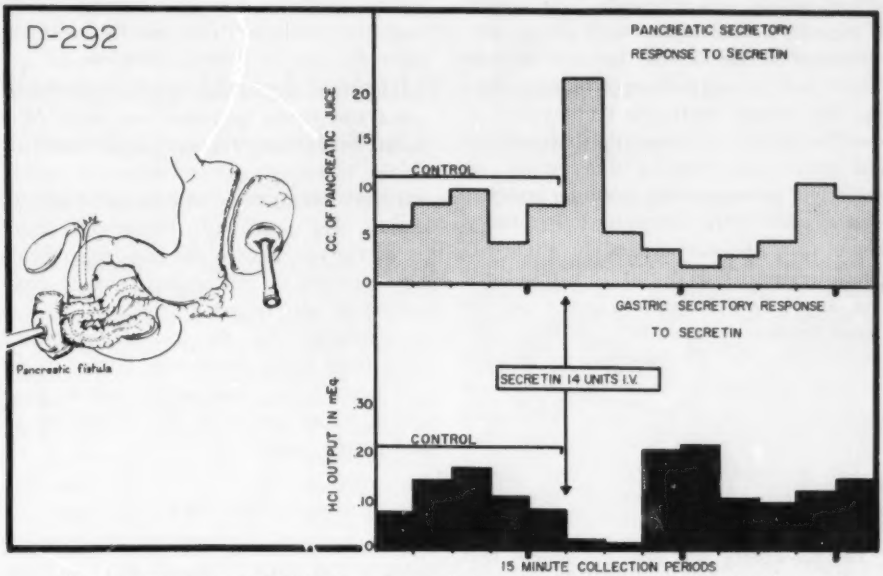


Fig. 5.—Effect of single injection of secretin in pancreatic fistula and Heidenhain pouch dog. The intravenous injection of secretin results in an immediate rise in the output of pancreatic juice and a concomitant inhibition of the gastric secretory response.

following the construction of the total pancreatic fistula might in some may be secondary to a metabolic disturbance. However, serum electrolytes were maintained within normal limits through appropriate intravenous injection of Ringer's solution, ⅙ M

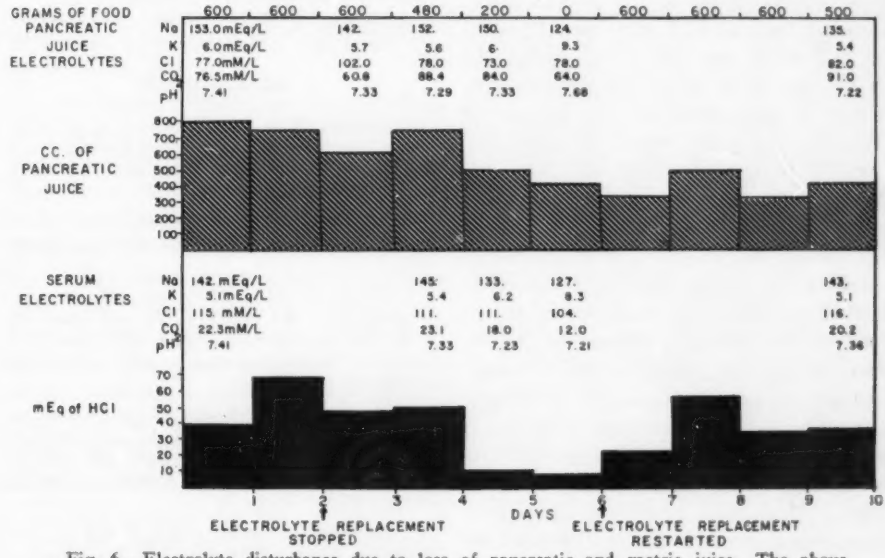


Fig. 6.—Electrolyte disturbance due to loss of pancreatic and gastric juice. The above Figure illustrates the metabolic acidosis which results when appropriate parenteral electrolyte replacement is not given. Note particularly the reduction of Heidenhain pouch output during the acidotic phase.

sodium lactate, and potassium. The amount of parenteral fluid replacement was roughly equivalent to the amount lost via the pancreatic and gastric fistulae, urine, etc. One dog was denied electrolyte therapy and allowed to become progressively more acidotic and dehydrated. During this period, the electrolyte concentrations in serum and pancreatic juice were determined frequently. These data are summarized in Figure 6. There was no rise in output of hydrochloric acid from the Heidenhain pouch as the animal became more acidotic; in fact, a decrease occurred. The decrease is at least partially explained by the reduction in food intake as the dog became more dehydrated and acidotic. It seems safe to conclude that in this preparation the Heidenhain pouch does not respond to acidosis by hypersecretion of acid gastric juice.

The possibility was also considered that the amount of parenteral fluid therapy given to the pancreatic fistula dogs might in some way stimulate the gastric secretory output. An identical volume of similar parenteral fluids was given to a controlled Heidenhain pouch dog. There was only a slight increase in the gastric secretion from a control level of 17.7 mEq. per 24 hours to 20.2 mEq. per 24 hours during the 10-day interval of parenteral fluid therapy. It must be remembered that this animal was relatively overloaded because the fluid loss from the Heidenhain pouch alone was one-third the loss of that in the Heidenhain pouch pancreatic fistula dogs. This suggests that the increase in gastric secretory output following the construction of a pancreatic fistula is not adequately explained by alterations in the acid-base balance or by replacement of the parenteral fluid therapy necessary to maintain the animal in balance.

Previous work done in this laboratory indicates that total pancreatectomy has little or no effect on the gastric secretory output from a Heidenhain pouch.[5] Further corroborating evidence is the low incidence of ulcers in the Dragstedt series following total pancreatectomy. These experiments indicate that the loss of pancreatic juice from the duodenum does not by itself explain the rise in gastric secretion.

It is possible that creation of the external pancreatic fistula provoked secretion of a gastric stimulating humoral agent resulting in the increased gastric secretory output. Further investigation will be necessary to explore this possibility. Postmortem studies of the pancreas in the pancreatic fistula dogs revealed no morphologic change from normal in islet or acinar elements. It can be concluded that the loss of pancreatic juice from the gastrointestinal tract in the presence of morphologically normal pancreatic tissue results in the stimulation of acid gastric juice.

Summary

Total pancreatic fistulae were constructed in five previously controlled Heidenhain pouch dogs. The gastric secretory output increased in all dogs after construction of the pancreatic fistula, with two of the animals showing over a 400% increase. Several possible explanations for this increase in gastric secretion are discussed.

950 E. 59th St. (37).

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Emergency Mitral Valvular Commissurotomy Preceding a Second Aortic Embolectomy

ELLIOTT S. HURWITT, M.D.; PHILIP GLOTZER, M.D., and JOHN B. SCHWEDEL, M.D., New York

Embolization to the bifurcation of the abdominal aorta represents one of the more serious complications that may develop in a patient with rheumatic mitral stenosis. Unless prompt embolectomy is accomplished, the mortality rate is extremely high. The three survivals in the series of 11 consecutive operative cases of saddle embolus of aorta previously reported from this hospital [1] all were subjected to surgery within seven hours of the onset of embolization. With the widespread performance of surgical procedures for the mechanical relief of the obstructed mitral valve, one may now evaluate the management of saddle embolization with reference to one of three time categories: occurring prior to, during, or after mitral valvular commissurotomy. Each of the three survivals previously reported fell into a different one of these categories.

When aortic embolization occurs during the course of surgery on the mitral valve, prompt embolectomy is associated with a high degree of success. The patient is already anesthetized and on the operating table, and the unfavorable dynamic situation within the heart has been corrected. This complication occurred three times among the first 300 patients subjected to mitral valvular surgery at this hospital; aortic embolectomy was performed without delay, and all three of these patients are living and well at this time. Similarly, prompt operation should carry with it a fair degree of success when embolization occurs some time following the intracardiac procedure. When the mitral stenosis has not been corrected, however, and aortic embolization occurs, the problem arises whether emergency embolectomy is to be performed first and followed by elective commissurotomy, or if emergent opening of the mitral valve should precede the extraction of the embolus from the aorta. Although some patients have been managed successfully by the regimen of emergency embolectomy followed in several days, weeks, or months by mitral commissurotomy, the more usual course is that of recurrent morbidity and fatalities, due chiefly to repeated embolization, shock, or congestive failure; additional salvage might have resulted had the valvular obstruction been corrected.

Emergency mitral surgery preceding aortic embolectomy has been performed successfully on a 53-year-old woman. Embolization to the aortic bifurcation had recurred, despite adequate anticoagulant therapy, one week after initial aortic embolectomy. The details of the case follow.

Report of a Case

A 53-year-old Puerto Rican woman was admitted to the Montefiore Hospital on Oct. 21, 1958, with the chief complaint of progressive dyspnea of two weeks' duration. The patient had experienced an episode of acute rheumatic fever 21 years previously. A heart murmur was noted at that time, and she was advised to limit her activities. For the past 18 years she had noted progressive dyspnea on exertion. Eighteen years ago and four years prior to this admission she had received digitalis for a short period of time because of dyspnea. Intermittent paroxysmal nocturnal dyspnea had been present for several years, and currently she

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From the Surgical and Medical Divisions, The Montefiore Hospital.

experienced three-pillow orthopnea. Two weeks prior to admission the patient experienced an acute gastrointestinal upset, followed by progressive weakness and dyspnea. For three days she had been bedridden because of severe orthopnea, and she continued to complain of nausea. On admission the patient had atrial fibrillation with a rapid ventricular rate, with an apical rate of 144, and a peripheral pulse of about 80 per minute. Her neck veins were distended, and showers of moist rales were heard in both lung bases; the liver was not enlarged, and there was no ankle edema. The heart was enlarged, with the point of maximal impulse at the anterior axillary line in the fifth interspace. The pulmonic second sound was louder than the aortic, and the first sound at the apex was accentuated. There was a loud rumbling diastolic apical murmur.

The patient was considered to be in mild congestive failure with an uncontrolled atrial fibrillation and she was given a digitalis preparation orally; because she continued to vomit this was changed to the intravenous route. On the morning of her third hospital day she suddenly developed the classical signs of embolization to the bifurcation of the abdominal aorta. Embolectomy was performed under spinal anesthesia, within four hours of the embolization. A transperitoneal approach was employed, as described in the previous publication on this subject from this hospital,[1] and a large clot was removed from the bifurcation of the aorta and from both iliac arteries, with prompt restoration of the pulses in both legs. Anticoagulant therapy was instituted with heparin and continued with warfarin; the patient was ambulatory on the fifth postoperative day. A blood sample on that day showed a prothrombin time of 33.4 seconds, with a control time of 11.8 seconds. On the morning of the seventh postoperative day her prothrombin time was 23.6 seconds, while the control time was 11.8 seconds. Shortly after breakfast on the seventh postoperative day the patient again exhibited the clinical picture of embolization to the aortic bifurcation. Phytonadione (vitamin K_1) oxide was given intravenously, and the operative procedures were started within four hours of the onset of this acute episode. First, mitral valvular commissurotomy was accomplished under general anesthesia through the left auricular appendage. There was a tight mitral stenosis, with no insufficiency, and no thrombi were felt; a satisfactory opening of the valve was accomplished. After closure of the chest the previous laparotomy incision was opened, and a large completely obstructing embolus was removed through the previous aortotomy incision. The total operating time for the two procedures was 2 hours and 20 minutes. The patient received 500 cc. of blood during the operation, at the conclusion of which both femoral pulses were strong and her condition was good. Her postoperative course was complicated by a deep wound infection, a period of profound emotional disturbance, and negativism thought possibly to be related to small cerebral emboli, and one episode of massive gastrointestinal bleeding associated with anticoagulant therapy and requiring three units of blood for correction. However, she was ambulatory at time of discharge from the hospital on Dec. 11, 1958, and has continued to be well to date, with no evidence of circulatory insufficiency to her legs or neurological deficit and with profound relief of her presenting complaints at the time of her admission.

Comment

Six aortic embolectomies were performed, in five patients, from the time of our initial report [1] to the end of 1958. Three of the five patients are alive and well, including the subject of this report (Case 5); aortic embolization had occurred during commissurotomy in two, and the embolectomy had been preceded by emergency commissurotomy in the third. In the two patients who died aortic embolectomy had been the only surgical procedure, and

Consecutive Aortic Embolectomies, May, 1955—December, 1958

Case No.	Age	Sex	Etiology	Date of Operation	Hours to Surgery	Previous Emboli	Cardiac Rhythm	Result
1.	42	F	During mitral commissurotomy	6/27/56	6	No	A.F.	Excellent; still well
2.	32	M	Mitral stenosis	2/21/57	16	No	A.F.	Died 3/1/57; embolism (?); congestive failure
3.	67	F	Mitral stenosis	12/26/57	7	No	A.F.	Died 12/29/57; embolic shower
4.	49	F	During mitral commissurotomy	1/11/58	0	No	A.F.	Excellent; still well
5.	53	F	Mitral stenosis	10/23/58	4	No	A.F.	Emergency commissurotomy
				10/30/58	3½	Yes	A.F.	Excellent; still well

For 11 previous consecutive cases, see Ref. 1.

the stenotic mitral valve had not been corrected.

One of these patients (Case 2, Table), who was admitted to the hospital as an emergency with aortic embolization just two weeks after he had been advised to enter the hospital for elective mitral valvular surgery, lived for eight days with good restoration of circulation to his lower extremities following embolectomy, and should reasonably have been expected to survive. In retrospect mitral valvular commissurotomy, performed as an emergency before the embolectomy, might have prevented the sudden terminal episode (possibly embolic) and the stormy postoperative course. Case 3 represents a potentially similar situation. Fatalities following aortic embolectomy have occasionally been associated in the past with failure to correct the stenotic mitral valve. One of us (E. S. H.)[2] encountered such an experience in 1949. Although a satisfactory embolectomy had been accomplished within five and one-half hours of embolization in a 48-year-old man, acute heart failure developed seven hours postoperatively. The autopsy showed an extreme degree of mitral stenosis, in addition to which the left auricular appendage contained a large firmly organized thrombus. Gumbiner and Keeley[3] reported a fatality in a patient following aortic embolectomy, in whom the autopsy disclosed tight mitral stenosis and a pedunculated thrombus in the left auricle.

The first report describing emergency mitral valvulotomy before aortic embolectomy is that of Glenn and McNeill[4] in 1957. Because of similar experiences with deaths due to unrelieved mitral stenosis following aortic embolectomy, these authors reasoned that the results in the management of saddle embolism should be better with the combined procedure of commissurotomy-embolectomy than had previously been accomplished with a policy of performing embolectomy only. In addition, emboli dislodged during the commissurotomy might be expected to be arrested at the site of aortic obstruction and be removed with the subsequent aortic embolectomy. Furthermore, lower limb survival should be enhanced with the improved cardiac output following a satisfactory valvulotomy. Glenn applied this reasoning with success in the case of a 47-year-old woman with severe congestive heart failure, performing first the mitral commissurotomy and then the embolectomy. One may add a previous embolectomy as an additional indication for the combined procedure. Shumacker[5] successfully performed mitral commissurotomy as an emergency immediately preceding aortic embolectomy in a patient who had undergone left common iliac embolectomy one week previously at another hospital.

Only one successful repeated saddle embolectomy has been encountered, reported by Thomas et al.[6] in 1957. In contrast to the present instance, their case recurred three and one-half months after the initial embolectomy; the patient was not receiving anticoagulants; and the aorta itself was incised only once, during the second episode, the other clots being removed through iliac and femoral arteriotomies.

The subject of the present report is apparently the second known case to survive two aortic embolectomies, and the third reported case in which a patient with aortic embolization was treated with combined commissurotomy-embolectomy. On the basis of these experiences, the procedure of emergency mitral valvular commissurotomy preceding aortic embolectomy merits serious consideration in patients with embolization to the aortic bifurcation who have rheumatic mitral stenosis.

Summary and Conclusion

Emergency mitral surgery preceding aortic embolectomy has been performed successfully on a 53-year-old woman. Embolization to the aortic bifurcation had recurred, despite anticoagulant therapy, one week after initial embolectomy.

This is the second known case to survive two aortic embolectomies, and the third

reported instance in which a patient with aortic embolization was treated with combined commissurotomy-embolectomy.

Emergency mitral commissurotomy preceding aortic embolectomy in patients with mitral stenosis merits favorable consideration.

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Clinical and Pathological Observations in Three Cases of Ainhum

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Ainhum is a disease seen primarily in the Negro race and is characterized by progressive constriction of the base of one or more toes. The disease is more prevalent in tropic and semitropic areas,[8,10] the majority of patients having been reported from Africa and Central and South America.[5,8] Since Clarke's [2] original report in 1860, it has remained a medical curiosity, particularly in the United States. Only 71 patients have been reported in this country since Hornaday's [17] first published case in 1881. Forty-two of these were summarized by Spinzig [10] in 1939, ten by Vaughn, Howser, and Shropshear [14] in 1945, and ten by Hucherson [5] in 1950.

Because of the infrequency of the disease in this country, the three patients with ainhum who have been seen at The Johns Hopkins Hospital between 1931 and 1958 are deemed of sufficient interest to report.

Report of Cases

CASE 1.—A 43-year-old Negro woman was seen in The Johns Hopkins Hospital outpatient department during July, 1931, because of a painful right fifth toe. Occasional tenderness in the toe had been present for one year. Two months previously she had struck the toe, and an increase in pain resulted for which she sought medical advice. Examination revealed a deep fissure almost encircling the toe at the distal interphalangeal joint. Superficial infection was noted in the region of the fissure. The blood Wassermann reaction was negative.

She was next seen two years later, at which time the constriction at the distal interphalangeal joint had deepened. Posterior tibial and dorsalis pedis pulses were palpable. X-ray revealed a deep fissure in the region of the distal interphalangeal joint with osteolysis of the underlying bone. The toe was amputated in June, 1933. Complete relief of pain resulted. No evidence of involvement of other toes was present when she was last seen in 1942 for treatment of lichenoid dermatitis of the neck and hands.

CASE 2.—A 29-year-old Negro woman was seen during February, 1946, complaining of pain in the left fifth toe. Past history revealed that she had been treated for syphilis and pelvic inflammatory disease. Three years previously she had developed a corn on the affected toe, which she had trimmed frequently. Superficial infection had been present intermittently. Four months previously she had noted the onset of pain in the toe.

Examination revealed that the left fifth toe was attached to the foot by only a small pedicle. The toe was tender on the dorsal and ventral surfaces, although no evidence of acute infection was present. The dorsalis pedis and posterior tibial

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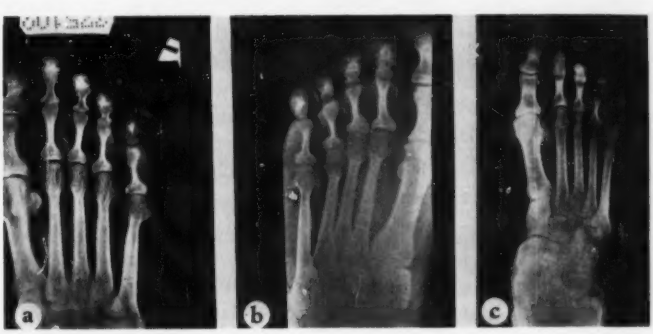

Fig. 1.—Roentgenograms taken shortly before amputation of the fifth toe. (*a*) Case 1. (*b*) Case 2. (*c*) Case 3. In each, constriction of the proximal portion of the fifth toe is present, with osteolysis of the underlying bone.

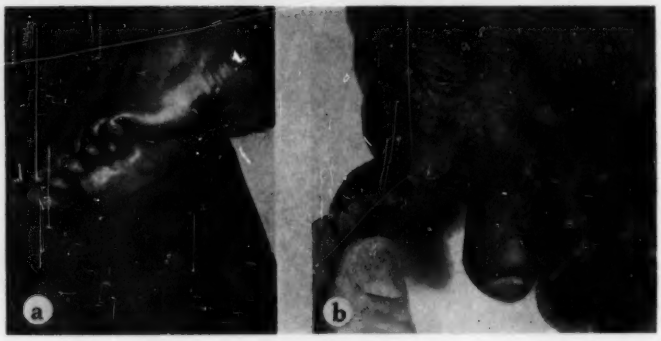

Fig. 2. — Photograph taken at the time of amputation of the fifth toe. (*a*) Case 2. (*b*) Case 3. The constricting groove can be seen at the proximal end of the fifth phalanx.

pulses were present bilaterally. Neurological examination was normal. The toe was amputated through the metatarsophalangeal joint, and complete relief of pain followed. Microscopic examination of the specimen revealed chronic inflammation and scarring. No evidence of involvement of other toes was noted when she was seen in the obstetrical clinic in 1949.

CASE 3.—A 62-year-old Negro was seen in The Johns Hopkins Hospital accident room on Dec. 3, 1955, because of pain in the right fifth toe. Ten days previously he had dropped an electric iron on the toe. His past health had been excellent, and no history of previous illness, operations, or injury was obtained. The family history was unremarkable. Examination revealed marked tenderness of the distal end of the toe. Several superficial fissures were present between the fourth and fifth toes. Oral temperature was normal. An x-ray of the foot was considered to be normal. A diagnosis of contusion of the right fifth toe with superimposed superficial infection was made. He received daily intramuscular injections of penicillin for four days, was advised to soak his foot in a dilute solution of potassium permanganate twice daily. Four weeks later he was asymptomatic and no evidence of infection was noted.

Almost two years later, on Jan. 3, 1958, he was again seen. He stated that although he had soaked his foot daily since last seen, his toe had gradually become worse. Actually the impetus for his return was the development of marked tenderness in the toe five days earlier. Examination revealed a constriction at the distal end of the proximal phalanx, so that the distal portion of the toe was attached by a pedicle measuring 7 mm. in diameter. Passive movement of the toe produced moderately severe pain. Sensation was normal to pin and cotton, and proprioception was intact. The temperature of the distal portion was grossly equal to that of the other toes. The dorsalis pedis and posterior tibial pulses were present bilaterally. No keloids were present on the body. X-ray revealed a constriction of the proximal portion of the right fifth toe with osteolysis of a portion of the proximal and distal phalanges. The soft tissues of the distal portion were swollen. The x-rays obtained in December, 1955, were reviewed, and it was apparent in retrospect that a deep fissure had been present at the time of that examination on the plantar surface of the toe in the region later involved by the constricting process. No evidence of bone change was noted. On Jan. 24, 1958, the right fifth toe was amputated through the pedicle. Complete relief of pain followed. Microscopic examination of the specimen revealed chronic inflammation.

Etiology and Clinical Picture

Many theories as to the etiology of ainhum have been proposed. These have incriminated syphilis,[15] trauma,[10] chronic infection,[10] and trophoneurosis.[16] In the three patients herein reported, a history of low-grade chronic infection was the only factor common to all. The second and third patients related the onset of the condition to minor trauma; no such suggested precipitating event was suggested in the first patient.

In these patients there was no apparent correlation with previous or coexisting disease. The first patient was later treated for lichenoid dermatitis of the neck and hands; the second patient had been treated previously for syphilis and pelvic inflammatory disease, while the third patient had always enjoyed good health.

Clinically, the onset was marked by the appearance of a transverse groove on the plantar surface of the toe in the region of

Patients with Ainhum Reported in the United States and Not Included in Previous Summaries

Author	Year	Age	Sex	Race	Toe Involved	Previous Trauma or Infection	Duration of Symptoms when Seen	Pathology
Martens & Norris[8]	1945	50	M	N	R-5th	Yes—laceration 4 yr. previously	4 yr.	Moderate hyperkeratosis and acanthosis; subcuticular round-cell infiltration; chronic osteomyelitis; essential lesion thought to be chronic osteomyelitis
Tye[12]	1946	77	M	N	L-5th	No	6-8 mo.	Stratum corneum thickened particularly near the point of amputation; numerous thin-walled vessels distal to point of constriction; decrease in amount of remaining bone
Shaffer[9]	1947	57	F	W	L-3rd L-4th	Corn with infection Corn	18 mo. 2 mo.	Epidermis hypertrophied at and distal to point of constriction; slight perivascular round-cell infiltration; simple atrophy of bone
Young[13]	1948	54	M	N	L-5th	Yes—laceration 15 yr. previously	15 yr.	Keratotic skin changes; generalized endarteritis; rarefying osteitis
Bender[1]	1950	57	M	N	R-5th L-5th	No No	4 yr. 6 mo.	Hyperkeratosis and parakeratosis at site of constriction Nonspecific fibrosis
Stack[11]	1950	32	M	N	R-5th	No	3 mo.	"Similar to those described by Spinzig in 1939"
Jacobs, Butz & Felts[6]	1951	25	M	N	R-5th L-5th	Yes—fungus infection of both feet 5 yr. previously	5 yr.	Hyperkeratosis in region of constriction; lymphocyte infiltration in perivascular and periosteal areas; mild periostitis, osteolysis
Chancey & Gipson[3]	1956	18	M	N	R-5th	Yes—infection of both feet 4 ½ mo. previously	4 ½ mo.	Hyperkeratosis and acanthosis of epithelium; osteolysis and periosteitis of bone in region of constriction
Burch & Hale[5]	1957	23	M	N	L-5th	Yes—laceration 11 yr. previously	11 yr.	Hyperkeratosis; low-grade chronic inflammation of digital arteries, periosteum, soft tissues, and skin
Riley & Cantrell	1959	43	F	N	R-5th	No	1 yr.	Not submitted for microscopic examination
	1959	29	F	N	L-5th	Yes—corn with infection	3 yr.	Chronic inflammation and scarring
	1959	62	M	N	R-5th	Yes—contusion 2 yr. previously	2 yr.	Chronic inflammation

the digitoplantar fold. This became circumferential and deepened until the bulbous distal portion of the toe was attached by a thin pedicle. Osteolysis of the phalanx occurred in the constricted area. Neurological examination was normal and skin temperature of the toe unchanged. No evidence of peripheral vascular disease was noted.

All were prompted to seek medical advice because of the onset of pain. In all three cases the disease was progressive and eventually required amputation.

Pathology

The amputated toe was submitted for microscopic examination in two of the three cases reported above. In each, the predominant feature was chronic inflammation. No bands of hypertrophied fibrous tissue were noted.

There is little agreement in the literature as to the essential pathologic lesion. Spinzig [10] noted hypertrophy of the epidermis in the vicinity of the constriction and a rarefying osteitis involving the phalanx. Others [7] have felt that the epithelial changes seen are not striking. In each of the three patients herein reported, thickening of the epidermis in the region of the fissure was apparent clinically, and it was confirmed by microscopic examination in the two specimens submitted to the surgical pathology laboratory.

Martens and Norris [8] have suggested that the essential lesion may be a chronic osteomyelitis and that the constriction is the result rather than the cause of the bony changes. Although careful histologic examination of the bone was not done, the presence in our third patient of a deep fissure without bone change on x-ray indicates that the process was not primary in the bone in this instance.

Between 1945 and 1957, nine additional patients have been reported who are not included in the previously mentioned summaries. These patients and the three reported herein are included in the Table with a brief summary of the pathologic features noted.

Summary

Three patients with ainhum were seen at The Johns Hopkins Hospital between 1930 and 1958. The following features were noted:

1. In each a history of chronic low grade infection of the toe was obtained.

2. Each was prompted to seek medical advice because of the onset of pain in the toe, which was relieved by amputation.

3. Microscopic examination of two of the amputated toes revealed chronic inflammation.

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Spontaneous Perforation of the Common Duct

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Rupture of the extrahepatic bile ducts should be considered in the differential diagnosis of bile peritonitis.

The following entities have been incriminated in bile duct perforation: (1) previous biliary tract surgery [3,6,7,14,16]; (2) abdominal trauma [1,10,12,15]; (3) vascular insufficiency with necrosis of the duct wall [9]; (4) choledochal diverticulum [2]; (5) erosion by tumor [7]; (6) acute pancreatitis.[10,17] There is a further group of patients in whom the cause of perforation is unknown. This idiopathic group has been reported most frequently in early infancy and beyond middle age.[8,11,13]

At the Philadelphia General Hospital we have recently seen two patients with bile peritonitis due to spontaneous perforation of the common bile duct.

Report of Cases

CASE 1.—A 55-year-old Negro was admitted Nov. 7, 1956, from a local prison with abdominal "fullness" of three weeks' duration. He had lost 40 lb. during the preceding six months. There was associated anorexia during this period. He also complained of intermittent lower abdominal pain of two years' duration. He was an alcoholic.

On examination, the patient appeared seriously ill. His pulse rate was 120 per minute. His blood pressure was 140/110 mm. Hg. Skin color was ashen. The abdomen was distended and diffusely tender. Peristaltic sounds were absent.

A diagnosis of peritonitis from undetermined cause was made. Supportive therapy was instituted, including transfusion of 500 cc. of blood and administration of intravenous saline solution. He deteriorated rapidly despite the supportive treatment and died before he could be prepared for laparotomy.

At autopsy, 2,000 cc. of green syrupy fluid was found in the peritoneal cavity. There were fibrinous adhesions in the right upper quadrant. As these were separated, greenish-black bile was expressed from a tiny defect in the anterior wall of the common duct just above the duodenum. The common duct was dilated.

A firm mass, which measured 2.5×2.0×1.0 cm., occupied the head of the pancreas, partially obstructing the common and pancreatic ducts. In the common duct proximal to the obstruction, a defect communicated with a small bile-filled cavity in the pancreas. The common duct was thickened and contained numerous mucosal ulcerations. The gallbladder was thickened and fibrotic and contained green sandy material.

Microscopically, the pancreas revealed organized and recent arterial thrombi. There was extensive necrosis associated with adenocarcinoma. The duodenum and common duct also revealed necrotic areas. The small perforations had occurred through necrotic areas. There was no direct invasion by tumor in the areas of perforation.

Comment.—The perforation in this patient probably resulted from impairment of the blood supply to the distal common duct due to the carcinoma arising in the head of the pancreas.

CASE 2.—A 76-year-old white woman was admitted to the Philadelphia General Hospital on Nov. 5, 1958. She was reported to have undergone a cholecystostomy at another institution three months previously. The patient stated that she wore a drainage tube for 25 days following her previous operation, and that two weeks after the tube had been removed she had experienced a bout of abdominal pain and transient jaundice. She noted the onset of colicky pain beginning Nov. 1, 1958. The pain radiated into her back and was associated with nausea and bile-stained vomitus. After a brief remission, her pain became worse and vomiting became intractable.

On the day following her admission, it was learned that the patient had undergone cholecystectomy and choledochostomy rather than cholecystostomy.

Physical examination on admission revealed an obese woman in acute distress, with tachypnea. Her sclerae were icteric. Her skin and mucous membranes were dry. Temperature was 100 F. The pulse rate was 130 per minute. Respiratory rate was 30 to 40 per minute. There was dullness over the right lung posteriorly and there were decreased breath sounds. Abdominal examination revealed a right upper quadrant operative scar.

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There was diffuse tenderness and diffuse rebound tenderness without muscle spasm. Direct tenderness was greatest in the mid epigastrium. Peristaltic sounds were absent.

The hemoglobin was 15:6 gm., and the white blood count was 6,300, with 90 polymorphonuclear cells. The serum amylase was 80 Somogyi units. Repeat amylase was 28 Somogyi units. Urinalysis revealed pyuria with bacteria. The urine was positive for bile. Electrocardiogram showed a sinus tachycardia at 130 beats per minute with nonspecific myocardial changes. The prothrombin time was 60% of normal. The blood urea nitrogen was 31 mg. %. Blood sugar was 200 mg. %.

Abdominal x-rays revealed an ileus pattern. The right diaphragm was elevated. There was a pleural reaction noted in the right hemithorax.

The patient was placed on gastric suction and was hydrated parenterally. Penicillin and streptomycin were given. This therapy caused immediate improvement. Shortly thereafter, however, she became lethargic and her abdominal tenderness increased. Abdominal paracentesis obtained bile stained fluid which contained Gram-negative rods on smear.

The patient was explored and the presence of bile peritonitis was comfirmed at laparotomy. A perforation from which bile was leaking was noted on the anterior wall of the common duct below the level of the previously ligated cystic duct remnant. The sharp edge of a stone was palpated at the defect. The common duct was dilated to approximately 30 mm. in size, and was wide enough to admit a finger. A choledochostomy was performed and four large stones were extracted. A No. 8 Bakes dilator passed readily through the ampulla. The duct was irrigated with saline and a No. 26 T-tube was inserted in the common duct. The peritoneal cavity was lavaged with 1,500 cc. of saline. Penicillin and streptomycin were instilled into the peritoneal cavity.

The patient had a smooth postoperative course until the seventh postoperative day, when she developed severe acute chest pain and dyspnea while ambulating. An electrocardiogram was compatible with pulmonary embolus. She died eight hours later despite active supportive therapy. Permission for autopsy was not obtained.

Comment.—This is a case of a delayed perforation of the common duct occurring three months after cholecystectomy and choledochostomy. There appeared to be an erosion of the duct wall by the sharp edge of a retained common duct stone.

Abdominal paracentesis was valuable in establishing the preoperative diagnosis of a purulent bile peritonitis.

This patient apparently would have survived a common duct perforation were it not for the unfortunate complication of pulmonary embolus.

General Comment

The diagnosis of a perforated bile duct is usually made at operation or autopsy. It is rarely considered prior to surgery. This was true in our patients.

Treatment has been delayed in many of the patients reported in the literature as well as in our patients because the presenting complaints have been bizarre. The clinical picture of a spontaneously perforated bile duct is unusual because it does not ordinarily have a specific historical pattern. The history is misleading because of the variety of underlying causes of such perforations.

The disorder (because of its rarity) is usually overlooked in the differential diagnosis of an acute abdomen. Furthermore, the physical findings in bile peritonitis are not impressive unless the bile has become infected or contaminated with pancreatic ferments or gastroduodenal contents. Abdominal paracentesis has proved to be of value in the identification of bile peritonitis.

We believe that operative treatment in patients with bile-duct perforation can be successful if performed early. A localized bile-filled abscess should be drained when present. Whenever possible, the underlying cause of rupture should be corrected. This may entail the removal of a common duct stone, or the resection of a tumor mass. The common duct should be explored and drained.[3,5,11] Continuity should be restored if there has been disruption.[15] In the presence of free bile peritonitis, the peritoneal cavity should be lavaged of its contaminant.[4] In some cases, the intraperitoneal instillation of appropriate antibiotics is advisable.

Summary and Conclusions

Two patients with spontaneous common duct perforation are briefly described. The etiology is reviewed.

Perforation of the extrahepatic bile ducts should be considered in the evaluation of an acute abdomen. In the presence of bile peritonitis, this diagnosis must be ruled out at laparotomy.

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Retrograde Colonic Intussusception

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Credit for the original recognition of retrograde intussusception of the alimentary tract probably belongs to John Hunter,[1] who postulated that this could occur. At the time of presentation of his paper "On Introsusception" before the Society for the Improvement of Medical Knowledge, he had neither seen nor heard of such a condition. Akehurst[2] made a review of the world's literature of this condition up to 1955, and found 104 instances of retrograde intussusception of all types. Twelve were of the colocolic type. Subsequently two additional cases have been recorded in the literature.[3,4] The present report provides the 15th recorded instance of simple retrograde colocolic intussusception.

Occasionally the small bowel is invaginated into the stomach at the site of a gastroenterostomy opening. Also, a retrograde intussusception is often associated with a typical forward intussusception, thus forming the so-called compound intussusception. However, simple retrograde intussusception of the small bowel or of the colon is a surgical curiosity.

A previous instance of retrograde intussusception of the colon has been reported from the Mayo Clinic by Balfour.[5] That case was of particular interest in that the intussusception was reduced at the operating table and was seen to recur forcefully before the eyes of the surgeon. Approximately 200 patients having intestinal intussusception have been treated at the Mayo Clinic during the past 51 years. Balfour's case and the one here reported are the only instances of retrograde intussusception of the colon in that series.

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Report of Case

A 78-year-old white widow was admitted to a Rochester hospital at 8 p.m., March 19, 1958, with the complaint of severe cramping pain in the left lower part of the abdomen and associated nausea and vomiting. She had been entirely well until two days prior to admission, when she had arisen from bed at about 11 p.m. and had defecated forcefully a large, loose stool. This had been an unusual experience for her. In about one hour had come the onset of severe lower left abdominal cramping, which occurred in waves of about five minutes' duration with intervals of about one hour. This condition had persisted for 12 hours without change, but then gradually the episodes of pain had become more frequent and severe, and nausea had developed. Several hours prior to admission the patient had consulted her physician and had been admitted to a local hospital. Enemata had resulted in no return of solution and no relief of symptoms, and she had been referred to the Mayo Clinic.

The patient had been seen previously at the Mayo Clinic for unrelated complaints. On the occasion of a general examination in 1952, one physician had noted a mass in the right side of the abdomen, but roentgenographic examination of the colon had revealed only a dolichosigmoid.

When examined in the hospital in March, 1958, this elderly woman appeared acutely ill. Her blood pressure was 168 mm. of mercury systolic and 96 diastolic, the pulse rate 80 per minute, and the temperature 99 F. The skin and mucous membranes presented evidence of mild dehydration. Examination of the abdomen revealed marked tenderness limited to the left lower quadrant. A visible and palpable mass in the left lower quadrant measured about 20 cm. in diameter. It was smooth and tender, and over it the percussion note was flat. The rest of the abdomen was tympanic. Peristaltic sounds were infrequent. Findings from rectal examination were normal.

The leukocyte count was 15,400 per cubic millimeter of blood, and a differential smear showed 94% of the leukocytes to be of the polymorphonuclear type. Results of urinalysis were essentially normal. A roentgenogram of the abdomen gave evidence that the right and transverse colon was dilated and filled with gas. A circumscribed area of density in the left lower abdominal region seemed to coincide with the mass detected by physical examination (Fig. 1).

Fig. 1.—Arrows point to the palpable mass in the abdomen. Note dilation of the proximal colon.

A tentative diagnosis of volvulus of the sigmoid colon was made, and the patient was prepared for emergency operation.

The abdomen was entered through a transverse left abdominal incision. The right and transverse portions of colon were found to be greatly distended with gas. A mass was encountered on the left which proved to be a retrograde intussusception of the sigmoid colon into the midtransverse colon. The intussusception could not be reduced by manipulation, and therefore left hemicolectomy with end-to-end anastomosis of the transverse colon to the lower part of the sigmoid was performed. No colonic stoma was established. The patient made an uneventful recovery and was dismissed from the hospital on the 11th postoperative day.

Gross examination of the resected specimen revealed the head of the intussusceptum to be formed by a polypoid tumor measuring about 4 cm. in diameter and having a broad base (Fig. 2). Microscopic examination of multiple slides from this tumor showed it to be a typical benign adenomatous polyp.

Comment

In adults, intussusception occurs usually as an intestinal diverticulum or a polypoid tumor is propelled through the lumen of the intestine by peristaltic activity, pulling with it the intestinal wall to which it is attached. In 4 of the 12 cases of retrograde colocolic intussusception reported by Akehurst,[2] a tumor of the colon was involved directly. Antiperistalsis is said to occur throughout the intestinal tract, but there is little evidence for the normal occurrence of strong reverse peristalsis in the sigmoid colon of man. Nevertheless, the retrograde intussusception in the patient reported here might best be explained by the association

Fig. 2.—Resected specimen (left colon) showing the polypoid tumor which led the intussusception.

of a sigmoid polypoid tumor, redundancy of the sigmoid colon, and active reverse peristalsis, with the result that the tumor was swept retrogradely to lead the intussusception.

Summary

A case of retrograde intussusception of the sigmoid colon into the midtransverse colon is reported. Fourteen similar cases have been found in the medical literature. This instance and one reported previously are the only cases of this kind that have been encountered in the experience of the Mayo Clinic among aproximately 200 intussusceptions of all types.

Mayo Clinic.

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Aortic Dissection Due to Translumbar Aortography, with Recovery Following Surgery

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Renal and neurological complications are the commonest serious sequelae of abdominal aortography. It has become increasingly evident that in some cases these occasionally fatal complications have been brought about by aortic mural dissection following the injection of dye, with partial or complete occlusion of aortic branch vessels. That this sequence of events can occur was strongly suspected by Gaylis and Laws,[1] who produced medial dissection experimentally in human abdominal aortas. However, McAfee,[2] in a review of data on over 12,000 aortograms, was able to collect only two cases of dissection following aortography, both of which ended fatally. On the other hand, Wolfman and Boblitt[3] reported four fatal cases from a series of 302 aortograms. In two of these the dissection directly contributed to the death. Fineberg and his associates[4] described a case of gangrene of the large intestine believed to have resulted from this mechanism.

Since the roentgenographic appearance and the surgical specimen removed from a case of dissection of the aorta produced by aortography in our own experience so strikingly resembled the experimental dissections demonstrated by Gaylis and Laws, documentation of this case seems to be justified.

Report of a Case

A 43-year-old white male welder was admitted to the Veterans Administration Hospital, Oteen, N.C., on Dec. 18, 1958, with a chief complaint of pain in the right foot and leg of nine weeks' duration. There had been no history of claudication, but he was unable to bear weight on this extremity. At the referring hospital, a diagnosis of arteriosclerosis obliterans was made, and a trial of conservative therapy, including oscillating bed and Buerger's exercises, was undertaken. There was symptomatic improvement, but pain at rest on the right continued.

One year before, the patient had had an episode of pain and numbness of the left lower extremity associated with low back pain, following a fall. He continued working until the onset of the present illness.

On examination at this hospital, the right foot was cooler than the left, with moderate rubor on dependency. The right calf and thigh were moderately atrophic. Deep tendon reflexes were normal. Right femoral pulsations were readily palpable but pulses distal to this were absent. On the left no pulsations were palpable. The patient could walk with the aid of a crutch.

Roentgenograms showed marked calcification of the abdominal aorta and both common iliac arteries. Chest x-ray was within normal limits. Laboratory examinations were also within normal limits.

Translumbar aortography was carried out on Dec. 23, 1958, under general anesthesia. A thin-walled 18-gauge needle with a short bevel was inserted at the level of the third lumbar vertebra and a good pulsatile flow of blood obtained. A 5 cc. test dose of 70% sodium acetrizoate (Urokon) was injected and a film taken (Fig. 1, *A*). Since this showed that the needle was in good position, 25 cc. of dye was rapidly injected by hand syringe and multiple exposures were made on the manual cassette changer (Fig. 1, *B*).

Several hours after this procedure, the patient complained of right back pain radiating to both legs, especially to the right leg. Over the subsequent few days he noted marked progressive weakness of the right lower extremity. There was an almost complete flaccid paralysis of the right leg. He was unable to get out of bed. There was obvious atrophy of the muscles of the thigh and calf. Sensation was diminished through all modalities, especially to pin prick in the L3-L4

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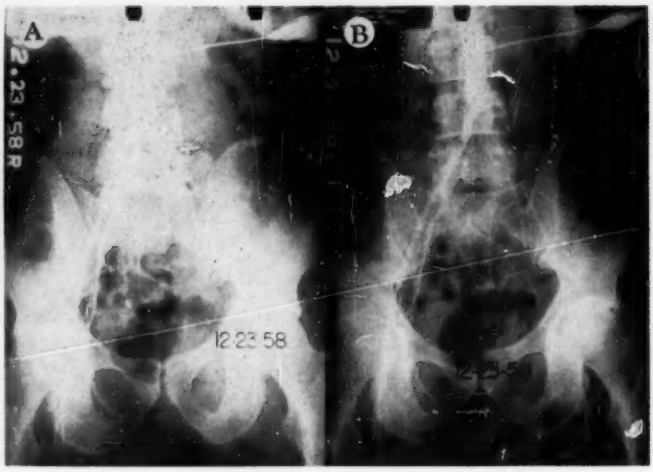


Fig. 1.—Lumbar Aortograms. *A*, test injection, 5 cc. of 70% sodium acetrizoate. The needle is opposite L3. There is no evidence of malposition of the needle or of mural injection. *B*, definitive injection, 25 cc. of 70% sodium acetrizoate. The aortic mural dissection is clearly outlined. The distal end of the pocket of dye is just above the aortic bifurcation, opposite L4.

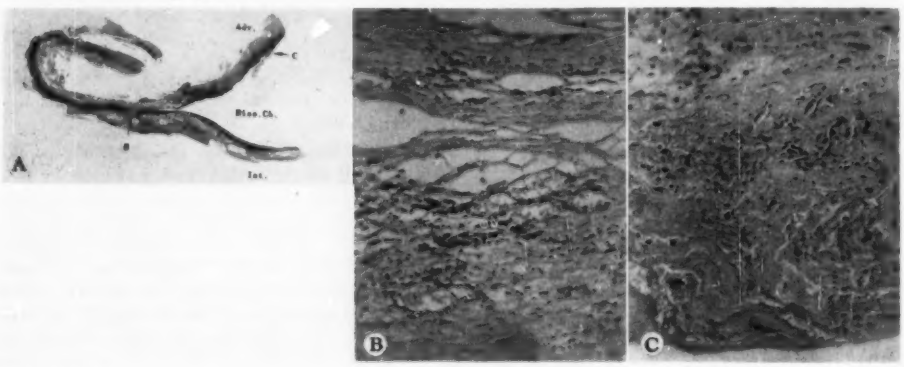
areas of the right thigh. Vibratory perception and light touch were diminished in the right thigh but normal below the knee. The sphincter tone was normal.

Spinal puncture and myelogram done five days after aortography were normal. However, 15 seconds after initiation of bilateral jugular compression during the lumbar puncture, the patient developed generalized convulsions lasting 30 seconds. Digital occlusion of the left cervical vessels produced no subjective or objective changes, while compression on the right after 30 seconds produced blurring of vision in the right eye, a sensation of faintness, and definite dysarthria. A femoral arteriogram on the right showed multiple filling defects in the superficial femoral artery.

It was thought that the patient had extensive vascular occlusive disease with a dissecting abdominal aortic aneurysm. It was felt that the process was advancing. Therefore, operation was urgently advised.

At operation six days after aortography, the distal aorta, the bifurcation, and both common iliac arteries were found to be intensely sclerotic and rigid. The aorta was opened longitudinally on its anterior aspect, distal to the inferior mesenteric artery, with the intention of performing a bypass bifurcation graft procedure. A false lumen was immediately entered. It extended upward as far as the clamp across the aorta and involved over half of the aortic circumference. This was interpreted as the distal portion of a dissecting aneurysm. Accordingly, the aorta was transected distal to the inferior mesenteric artery, and the true and false lumina were joined by resecting a portion of the intimal-medial partition between the

Fig. 2.—*A*, cross section of the upper portion of the resected aorta; × 4. Adv., adventitia; Diss. Ch., dissected channel; Int., intima. *B*, degenerative changes and lymphocytic infiltration in the media of the undissected portion; hematoxylin and eosin; reduced about 40% from mag. × 85. *C*, organizing thrombus within dissected channel; hematoxylin and eosin; reduced about 40% from mag. × 85.



two. The aortic bifurcation was resected and a Teflon bifurcation graft was anastomosed to the proximal end of the aorta and to the distal ends of the common iliac vessels bilaterally. Partial endarterectomy of the right common iliac artery was necessary to make this anastomosis possible. After exploration of the femoral and popliteal arteries on the right, it was decided that femoral popliteal bypass would not be possible owing to almost complete occlusion of the right popliteal artery.

The pathologic specimen consisted of the lower end of the aorta with the bifurcation and the first portion of each common iliac artery. The wall of the aorta revealed a separation of the media which involved nearly the whole circumference at the upper limit of the specimen (Fig. 2, *A*). Two lower lumbar arteries had been separated at their origins. Their lumina contained some soft clot which fell away during processing. Microscopically, the media had been split at about the junction of the outer one-third and the inner two-thirds. At the limit of the dissection the tissue was loose and showed some lymphocytic infiltration (Fig. 2, *B*). Beyond this, there were early degenerative changes and more lymphocytic reaction. This was consistent with the medial degeneration responsible for the dissecting aneurysm. Within the dissected channel there was a thin thrombus which was organizing (Fig. 2, *C*). The degree of organization was that which one would expect in one week, the actual time between aortography and resection. The intima of the undissected portion showed typical arteriosclerotic plaques.

The patient made a slow recovery. All pulses returned on the left, while those below the femoral on the right were not palpable. There was still mild rubor on the right on dependency, but both feet were warm. Sensation also improved in the right lower extremity. Three months later the patient was seen again. There was still moderate discomfort in the right foot and leg and ambulation was still limited. Rubor on dependency was still present. Knee jerk was now present on the right but was weaker than that on the left. Otherwise the patient appeared to be well.

Comment

The mechanism postulated and demonstrated experimentally by Gaylis and Laws appears to have been responsible for the dissection which occurred in the aortic wall of our patient (Fig. 4). This may also occur after carotid arteriography.[8] Apparently, if diseased wall opposite the site of arterial or aortic puncture is partly penetrated by the bevel of the needle, a good flow of blood may yet be obtained through that portion of the lumen of the needle which is in communication with the true lumen of the vessel. A small test injection of dye may fail to indicate malposition of the needle point. (This was the case in both Fineberg's and our aortogram, and in those reported by Wolfman and Boblitt.) Yet when 25 or 30 cc. of dye is rapidly injected, preexisting degeneration of the media allows separation and dissection of this layer of the vessel to occur. If the injection pressure exceeds intraluminal pressure, even a minimal initial split in the wall of the vessel created by the contrast substance will result in the spreading of the layers of the media and inclusion within the enlarging space of more and more of the bevel of the needle. Thus, toward the end of injection, an ever larger share of the dye will enter the dissected space. Temporary partial or complete occlusion of the carotid artery in arteriography or of the branches of the aorta in aortography may result much as in a spontaneous dissection. Whether the occlusion per se causes

Fig. 3.—Diagrammatic representation of probable mechanism of production of aortic mural dissection by lumbar aortography (see text). Note torn lumbar aortic branches on the right side. The dissection occurred opposite the site of aortic puncture by the needle.

pathologic changes due to ischemia peripherally, or by producing transient stasis of blood flow enhances the toxic effects of dye which has already traversed the occluded vessel is difficult to ascertain. Gaylis and Laws favor the latter theory.

While it is true that neurological symptoms referable to the right lower extremity were present in this patient before aortography, immediately afterward these became much severer. It is presumed that occlusion of lumbar aortic branches or of intercostal arteries occurred due to dissection of the media by the radiopaque dye. Two large lumbar arteries which were apparently torn at their origin by the dissection of the media were found in the gross specimen.

In all of the reported cases of dissecting aneurysm following aortography, the injection was made below the renal vessels. Current opinions regarding the optimal site of aortic puncture for lumbar aortography differ. Some authorities advocate puncturing high, at the level of the 12th thoracic vertebra. This avoids the most commonly diseased segment of the lumbar aorta (that below the level of the renal arteries) as well as the renal vessels, the superior mesenteric artery, and the celiac axis. Proximal to this level, atherosclerosis is rarely as severe as it is distal to the renals. This technique has the disadvantage of dissipation of dye in the visceral vessels, with correspondingly less concentration of contrast medium in the distal lumbar aorta, and in the iliac and femoral vessels. It also subjects the kidneys to the toxic effects of the dye. Others therefore advocate puncturing the abdominal aorta at the level of the third lumbar vertebra, in patients in whom the kidneys need not be outlined. This technique allows excellent concentration of dye where it is most needed. However, it carries with it the drawback of subjecting the most severely diseased segment of the abdominal aorta to the trauma of needle puncture and dye injection. Indeed the aorta is occasionally completely thrombosed at this level so that no aortogram is obtainable.

A technique combining the virtues of both high and low aortic puncture is that reported by Bruwer and Ellis.[6] In this technique, an initial rapid injection of a small amount of dye is made at the 12th thoracic vertebral level. This outlines the abdominal aorta and visceral branches. The latter can now be avoided by a second needle, placed well below the renals. Through this, a much larger quantity of dye, slowly injected, can opacify the aortic bifurcation, and the iliac, femoral, and popliteal arteries. If the initial high injection indicates marked disease or thrombosis of the abdominal aorta below the renal arteries, a second injection is not made. These precautions, coupled with recognition of the roentgenographic pattern of aortic mural dissection on the initial test film, may help to prevent the occurrence of more serious complications of mural dissection.

Intramural injections of dye at translumbar aortography occur rather commonly without causing serious symptoms, according to McAfee, and Wolfman and Boblitt. On the other hand, the presence of a true dissection found either at surgery or at autopsy is rare. Medial degeneration is probably necessary before such a dissection can occur.

Summary

A case of aortic dissection due to translumbar aortography is reported. The dissection was recognized roentgenographically, and the dissected area of lumbar aorta with the aortic bifurcation and both common iliac arteries was resected and replaced with a Teflon bifurcation graft. There was histologic evidence of medial degeneration in the undissected portion of the specimen. The patient recovered.

The mechanism of production of aortic medial dissection is discussed and methods of prevention are considered.

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Carcinoma of the Prostate with Metastases to the Testicle

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Secondary carcinoma of the testicle arising from carcinoma of the prostate has been reported only rarely in the literature. As far as we can ascertain, 26 cases [1-12] have been previously published. There are several possible explanations for the infrequent finding of these metastases.

1. The testicle is apparently rarely involved with metastatic tumors from any source. Helfert and Pinck [8] found fewer than 50 cases up to 1944. Klinger [10] reported 3 cases in 1951, and Price and Mostofi [9] in 1957 found 10 additional cases in the literature and added 38 of their own.
2. Possibly the testes removed as a therapeutic measure for prostatic carcinoma have not been examined histologically in some institutions.
3. Even where histological examination is a routine procedure, a small nodule could easily be overlooked.

It was thought worth while to report 2 cases of carcinoma of the prostate with metastases to the testicle because these will be the 27th and 28th cases to be described. It may be even discovered less frequently in the future, as the diagnosis of the primary lesion is being made much earlier than formerly and the testicles are being removed almost immediately. In fact, there has been some evidence [6] that the greater the length of the disease, the better the chance of such metastases occurring. From now on, it will possibly be found only in those patients who refuse orchiectomy.

Report of Cases *

CASE 1.—An 85-year-old white man was first admitted to the Morrisania City Hospital in Nov., 1946, complaining of urinary straining and frequency of three months' duration. Positive finding on physical examination was a 1+ prostate, hard, slightly irregular, and fixed, with residual urine of 200 cc. Remainder of examination was essentially negative. A clinical diagnosis of carcinoma of the prostate was made. Blood chemistry was normal with an alkaline phosphatase of 4.9 King-Armstrong units and an acid phosphatase of 1.5 Gutman units. An intravenous pyelogram was normal. An observation cystoscopy revealed a Grade 2 median lobe and a Grade 1 lateral lobe intraurethral intrusion with trabeculation of the bladder. Following cystoscopy, patient had a febrile reaction which required suprapubic cystostomy. After patient became afebrile, a transurethral resection of the prostate was performed on Dec. 23, 1946, and 11 gm. of tissue was removed. Histological report was moderate glandular hyperplasia with fragments of adenocarcinoma of the prostate. The patient was advised to have an orchiectomy, which he refused. He was placed on diethylstilbestrol 1 mg. t.i.d. and discharged to the Urological Out-Patient Department. He did not follow his hormonal treatment properly and he visited the clinic only occasionally. In November, 1953, he visited the Out-Patient Department, complaining of nocturia. He was told that an orchiectomy should be performed and this time he agreed to it. He was readmitted to the hospital on Nov. 25, 1953. Physical examination, including external genitalia, was essentially negative, except for a prostate enlarged one and a half times, hard, irregular, and fixed, with residual urine of 60 cc. Cystoscopy revealed a rigid, fixed irregular prostatic urethra with an adequate water way; bladder was trabeculated. CBC was within normal limits except for a lymphocytosis of 43%; urine negative; urea 28 mg. %; creatinine 1.95 mg. %; sugar 82 mg. %; acid phosphatase 2.5 Gutman units; alkaline phosphatase 14.0 King-Armstrong units. Chest x-ray was negative except for slight dilation of the thoracic aorta and hypertrophy of the left ventricle. Intravenous pyelography showed fair visualization with normal pyelograms. There was no evidence of metastases to spine or pelvis. A bilateral orchiectomy was performed on Dec. 4, 1953. The right testis was somewhat atrophic on gross examination. The left testis was surrounded with a hydrocele sac containing 3-4 cc. of clear

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From the Urological Service, Morrisania City Hospital, and the Urological Service, Lebanon Hospital.

* All microscopic sections were reviewed by Dr. I. M. Ratner, Associate Pathologist, Lebanon Hospital.

fluid. The parenchyma was markedly atrophic and showed replacement by fine granular tissue. On histological section, the right testis was atrophic; the left showed a well-differentiated adenocarcinoma of the same type as seen in the prostate previously.

CASE 2.—A 55-year-old man was admitted to Lebanon Hospital on May 23, 1950, because of a four-month history of frequency, difficulty voiding, nocturia (three to four times), dribbling, and a slow stream. The physical examination was essentially negative except for a 2+ enlarged prostate on rectal examination. The gland was regular and symmetrical, and no nodules were palpated. Cystoscopy disclosed a Grade 2 median lobe and lateral lobe enlargement. There was 2 oz. of residual urine. Intravenous pyelogram revealed essentially normal kidneys. On May 27, a transurethral resection of the prostate was performed and 20 gm. of tissue removed. Histological report was nodular hyperplasia with fragments of papillary adenocarcinoma (Fig. 1). The patient was placed on estrogenic therapy and discharged on June 2 ,1950. On July 17, 1955, he was readmitted because of a two-month history of discomfort in the left lower quadrant. For approximately one month he had noticed that the left testicle had become hard and painful. On physical examination, the left testicle was hard, smooth, and painful, but not enlarged. On July 19 a bilateral orchiectomy was performed. The right testicle was normal. The left was firm and on section showed many small whitish and well-circumscribed nodules, the largest measuring 0.5 cm. in diameter. Histological section (Fig. 2) revealed metastatic papillary adenocarcinoma of the left testis, showing a similar pattern to that previously reported in the prostate.

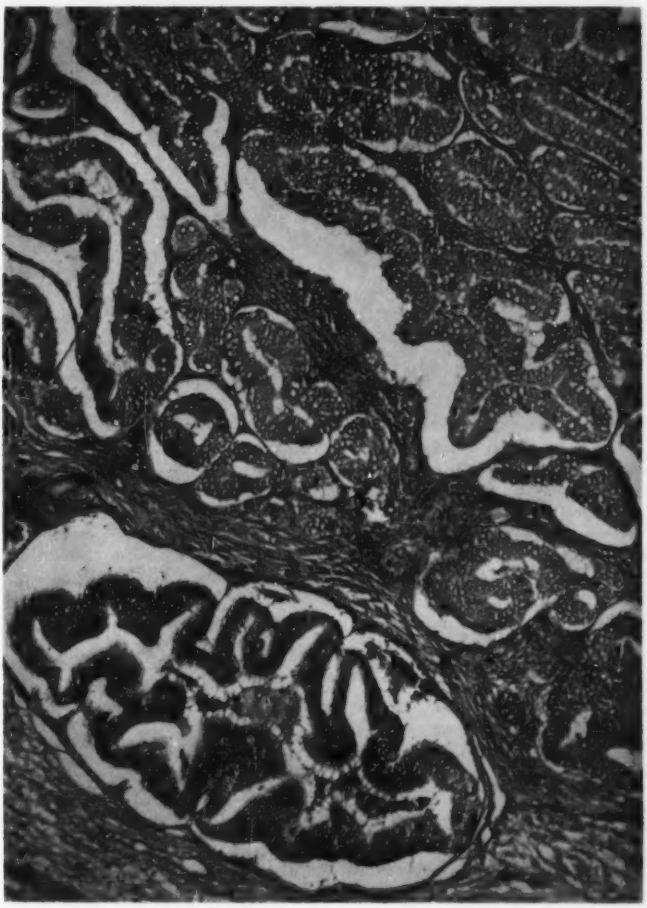

Fig. 1.—Papillary adenocarcinoma of the prostate.

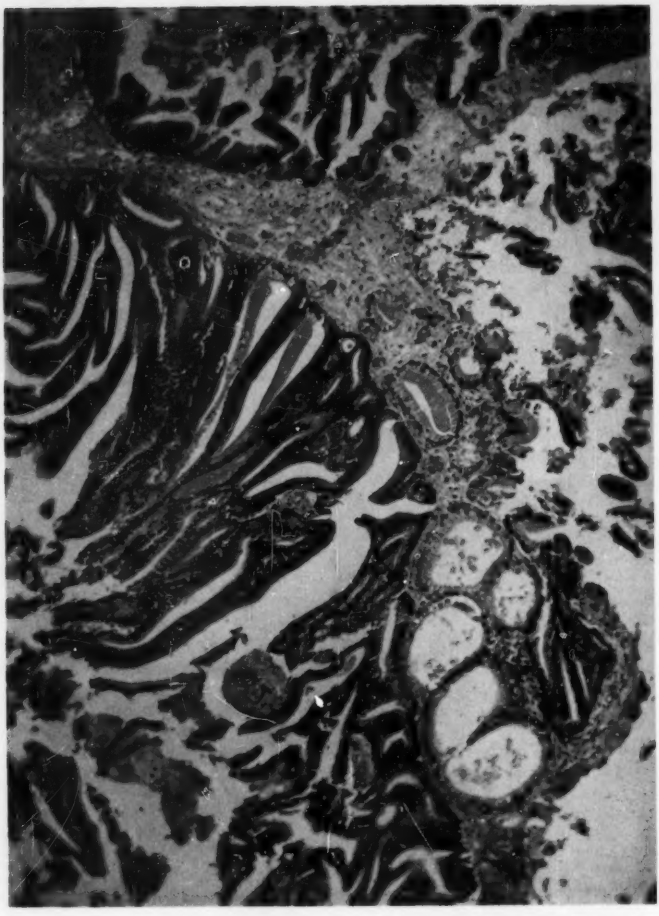

Fig. 2.—Section of testicle showing papillary adenocarcinoma histologically similar to Figure 1.

Comment

According to Willis,[14] the testis may become the seat of secondary tumors by any of three routes: (1) by retrograde venous extension or embolism; (2) by retrograde lymphatic extension; (3) by arterial embolism.

A fourth possibility is that suggested by Kay et al.,[6] by way of the lumen of the ejaculatory ducts to reach the epididymis and testis. He mentions this possibility because in his second case and in Bradham's[4] case tumor was demonstrable in the seminiferous tubules.

A fifth route of invasion could be by direct extension through the vas deferens but not within the lumen of the vas as exemplified by Thompson and Pilcher's[15] case, in which a tumor mass was found in the interscrotal portion of the vas deferens contiguous to the muscularis but not in the lumen. Howard, Hicks, and Scheldrup[10] have discussed all these possible routes of metastatic involvement of the testicle quite thoroughly. Our cases did not show any involvement of the cord with tumor, and we assumed that metastases occurred by way of arterial embolization.

Summary and Conclusion

The 27th and 28th cases of secondary carcinoma of the testicle arising from car-

cinoma of the prostate have been presented.

A discussion of the possible routes of invasion has been formulated.

It is suggested that, even though this lesion has occurred rarely in the past, it may be found even less frequently in the future.

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On the Drying Test of Gallbladder Bile

A New Method for Diagnosis of Gallstones

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I have emphasized for years that the presence of gallstones can be diagnosed by the form of crystallization after gallbladder bile is dried.[13-16] However, it became clear through later investigations [4,5,11,12,17] that this crystallization is closely related to the degree of inflammation of the gallbladder.

This report will present the details of the test and its clinical application, and some experimental studies of the test will be discussed.

It is very difficult to prove the presence of calculi in some cases of diseases of the biliary tract, especially in the case of a syndrome called cholecystopathy or choledochopathy. The customary means used at the present time to diagnose the presence of gallstones can be summarized as the following four: (1) history of present illness; (2) direct visualization by x-ray examination; (3) gall sand, gall mud, or cholesterol crystals obtained by duodenal drainage; (4) jaundice present or in the history.

The frequency of these findings in my department was as follows: Calculi were visualized by x-ray examination, as seen in Tables 1A and 1B, in only 47 out of 214 cases (22%), 33% of gallbladder stone, and 12% of common-duct stone. The correct diagnosis by sediment obtained by duodenal drainage was 23 out of 215 cases (10.7%) as shown in Table 2. The frequency of jaundice was 82.9% of common-duct stone, 43.4% of gallbladder stone, and 27.6% of cholecystitis without calculus (Table 3). Not only jaundice caused by obstruction of biliary passage by calculi, but hepatic jaundice also was included, and, furthermore, 27% of cases without calculus showed jaundice. Thus, it is emphasized that the value of jaundice in diagnosis of the presence of gallstones should not be overestimated.

As stated above, these findings were not fully satisfactory to prove the presence of calculi in all cases of cholelithiasis. The bile drying test to be presented in this paper will be a very simple and yet an effective aid for diagnosis of cholelithiasis,

TABLE 1B.—*Frequency of Visualization of Stone*

	No. of Cases	Stone	%
Gallbladder stone	99	33	33.3
Common-duct stone	113	14	12.4
Total	212	47	22.1

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TABLE 1A.—*Visualization of Stone by Cholangiography*

		Visualization of Gallbladder				Visualization of Gallbladder			
		Stone		Stone		Stone		Stone	
	No. of Cases	No. of Cases	%	No. of Cases	%	No. of Cases	%	No. of Cases	%
Gallbladder stone	99	60	60.6	10	10.1	6	6.1	23	23.2
Common-duct stone	113	98	86.7	8	7.0	1	0.9	6	5.4
Cholecystitis without stone	182	71	39.0	0	0	110	60.4	1	0.6

TABLE 2.—*Frequency of Gall Sand Proved by Duodenal Tube*

	No. of Cases	Sand	%
Gallbladder stone	85	10	11.7
Common-duct stone	130	13	10.0
Cholecystitis without stone	171	3	1.6

TABLE 3.—*Frequency of Jaundice*

	No. of Cases	Present, %	Jaundice in History, %
Gallbladder stone	115	22.6	43.4
Common-duct stone	170	68.2	82.9
Cholecystitis without stone	209	11.4	27.6

particularly when the stones are composed of bile pigment (bilirubin)* which comprises a high percentage of biliary calculi among the Japanese.

Method and Principle

Bile obtained from the gallbladder by needle aspiration during laparotomy is filtered, and 1 ml. of the bile is placed on a glass plate and dried in an incubator at 37 C for 24 hours.

A. Macroscopic Inspection

1. A regular pattern of crystals lined up radially is observed in most cases of cholecystitis with calcu[illegible] (Fig. 1, *A*).

2. N[illegible] crystal is observed in normal cases (Fig. 1, *B*).

3. No crystal or partial crystallization is observed in cases of cholecystitis without calculus.

B. Microscopic Examination

* "Bilirubin stone" contains less than 20% of cholesterol.

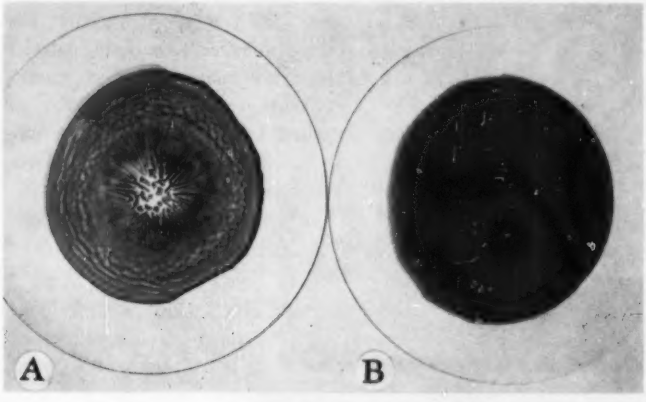

Fig. 1.—Macroscopic inspection of bile drying test. *A*, radial pattern of crystals. *B*, no crystals observed.

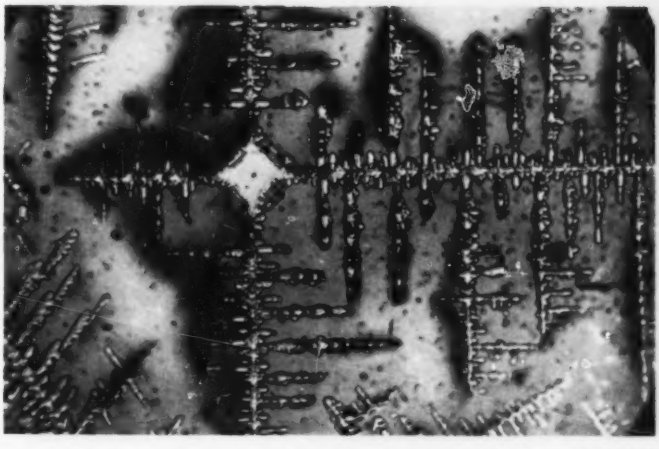

Fig. 2.—Fern-like structure.

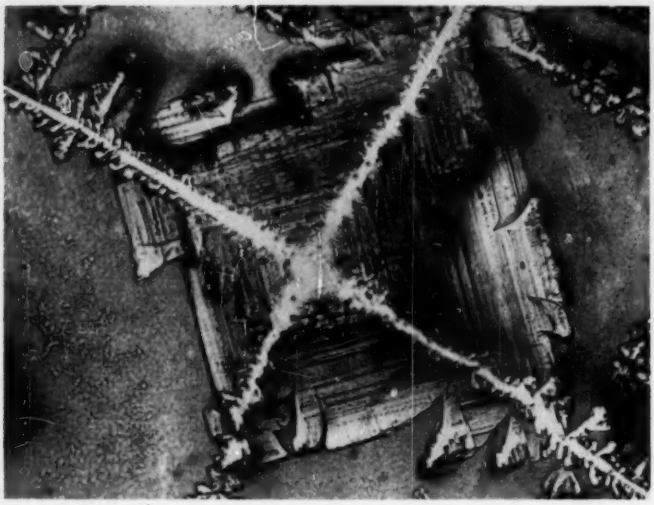

Fig. 3.—Crystal nucleus.

The dried bile material shows various patterns of crystals microscopically. These patterns will be classified as follows:

1. Typical crystals.
   (*a*) Fern-like structure (Fig. 2).
   (*b*) Crystal nucleus (Fig. 3).
2. Atypical crystals.
   (*a*) Thrum-like structure (Fig. 4).
   (*b*) Cross structure (Fig. 5).
   (*c*) Plate (Fig. 6).

These patterns are the same as those reported by Rydberg [22] and Grünberger [3] in the calculi found in the mucus from the uterine cervix.

To compare the microscopic classification and the macroscopic observation, the materials which microscopically show fern-like structure or a crystal nucleus are macroscopically observed as a radial pattern of crystals, and the materials which partly show crystallization are seen mostly as atypical crystals by microscopic examination. In some cases which did not show crystallization macroscopically atypical crystals were found on microscopic examination.

These observations are summarized in Table 4.

In 85 cases of cholecystitis with calculi, 68 cases showed typical crystals, 15 cases atypical crystals. In two no crystals were

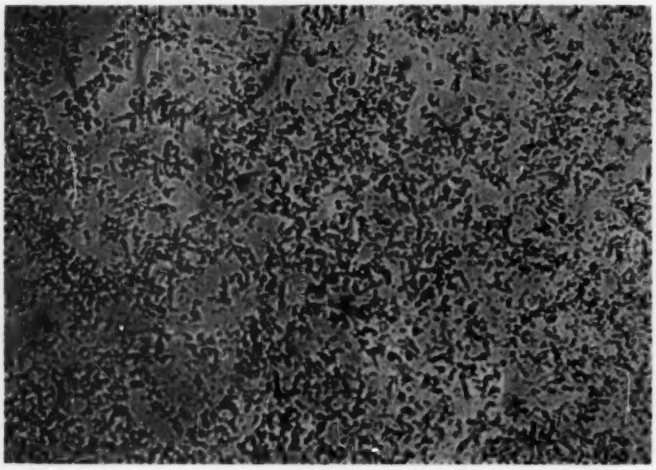

Fig. 4.—Thrum-like structure.

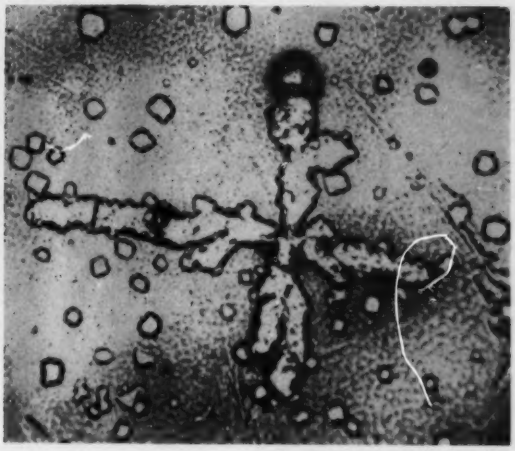

Fig. 5.—Cross structure.

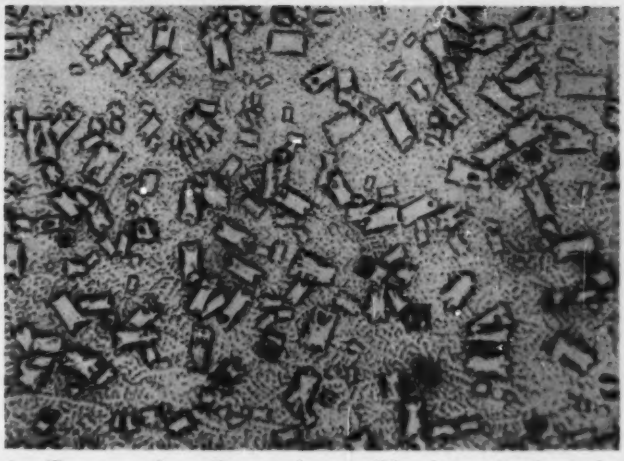

Fig. 6.—Plate.

seen. On the other hand, in 67 cases of cholecystitis without calculus, 4 cases (6%) showed typical crystals; 19 cases showed atypical crystals, and 44 cases were negative. In the four cases with typical crystals the operative findings seem to indicate that calculi might have existed and passed through the biliary tract.

TABLE 4.—*Results of Drying Test Shown by Microscopic Examination in 176 Cases*

	Typical	Atypical	Negative	No. of Cases
Normal biliary tract	0	0	24	24
Cholecystitis without stone	4	19	44	67
Cholecystitis with stone	68	15	2	85

It may be possible, from the facts described above, to diagnose the presence of biliary calculi when the dried gallbladder bile shows radial crystals macroscopically and typical crystals microscopically.

Clinical Application

According to the drying test of gallbladder bile obtained by puncture during surgery, there is a distinct difference between cases of cholecystitis with calculi, those without calculi, and normal cases, even though there are a few exceptions. Therefore, if it is possible to obtain gallbladder bile prior to surgery, it will be a great aid in diagnosis of gallstones. There might be a way to puncture a gallbladder per

cutem to obtain pure bladder bile, but this would be attended with serious risk and would also be technically difficult. Thus, "B" bile obtained through a duodenal tube is submitted for the test. A small amount of gastric juice or pancreatic juice mixed in the material does not interfere with the test, but if it is a large amount, or if peptone or magnesium sulfate is used as a cholekinetic, the crystallization will be interfered with. Therefore, pituitrin or atonin should be used to collect "B" bile. The bile is collected every 10 minutes until "C" bile is obtained and a typical portion of "B" bile must be used for the test to have clinical value.

The results were as follows: 1. Crystallization is rarely observed in cases where the biliary tract is normal. 2. Crystallization is mostly negative or atypical in cholecystitis without calculus. 3. Typical crystals are observed in most cases of cholecystitis with calculi.

Comment on Method

Two possible sources of error were found with the use of this test: 1. It was impossible to collect "B" bile for one or more of the following reasons: (*a*) empyema or hydrops vesicae felleae, (*b*) shrunk gallbladder, or (*c*) technical failure in collection. 2. The function of the gallbladder was restored or maintained even when calculus was present.

In the latter case, cholangiography would reveal a distinct visualization of the gallbladder and positive or negative visualization of calculi. Of course, if the calculi are visualized by x-ray examination, any indirect test such as this drying test would not be needed.

Origin of Crystal

The structures of these crystals are quite different, but all of them show positive Cl^- reaction negative $Ca^{\cdot}$ reaction, and the flame reaction indicates $Na^{\cdot}$. These crystals are therefore identified as sodium chloride, and it was also proved by means of x-ray diffraction (Debye-Scherrer method), as shown in Table 5.

TABLE 5.—*X-Ray Diffraction of Crystals (Debye-Scherrer Method)*

Diffraction Ring	Sample			NaCl by Other Investigators
	Bile 1	Bile 2	NaCl	
A	2.80	2.81	2.81	2.81
B	1.98	1.99	1.99	1.99
C	1.62	1.62	1.62	1.62,
D	1.40	1.40	1.41	1.41,
E	1.26	1.26	1.26	1.26,

Experimental Studies on the Relationship Between Crystals and Bile Elements

Normal gallbladder bile does not form crystals. However, when sodium chloride is added to it experimentally it starts to form typical crystals at the concentration of sodium chloride exceeding 600 mEq/L., while abnormal bladder bile forms typical crystals even when the concentration is as low as 100 mEq/L. From this fact, other factors besides the concentration of sodium chloride seem to participate in the crystallization, and it appears that crystallization is interfered with in the bile of normal persons or in cholecystitis without calculi, and this interference is decreased or lost in case of cholecystitis with calculi. Therefore, the metabolism of electrolytes, especially of sodium chloride, in bile was investigated.

Electrolytes.—Sodium and postassium were investigated by means of a flame photometer, and chlorine ion by the modified Furutani method.[2] As shown in Figures

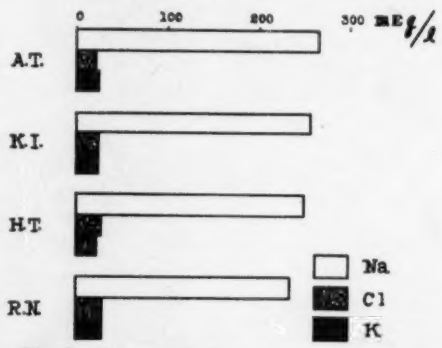

Fig. 7.—Amount of electrolytes in normal gallbladder bile of each case.

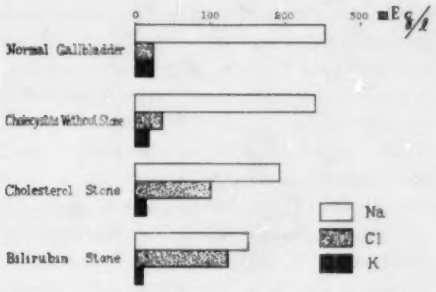

Fig. 8.—Amount of electrolytes in gallbladder bile in various conditions.

7 and 8, the concentration of chlorine ions in normal gallbladder bile was as low as 18.6 mEq/L. In case of calculi, especially of "bilirubin stone" (Figure 9), the chlorine concentration in gallbladder bile was mostly more than 100 mEq/L., which is similar to that of liver bile. In other words, the electrolytes in the bladder bile of "bilirubin stone" showed an increase of chlorine ion, and a decrease of sodium and potassium ions compared with those of normal bladder bile. At the same time, the drying test of bile showed typical electrolytes. On the other hand, in cases of "cholesterol stone" (Fig. 10) chlorine, sodium, and potassium ions showed various concentrations, and the drying test, accordingly, showed various results, negative to typical.

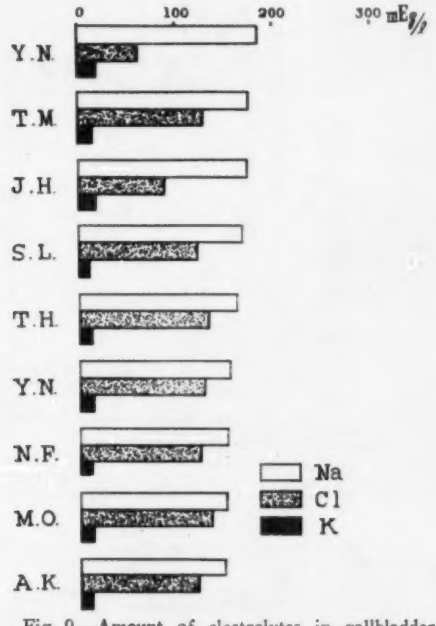

Fig. 9.—Amount of electrolytes in gallbladder bile of each case with bilirubin stone.

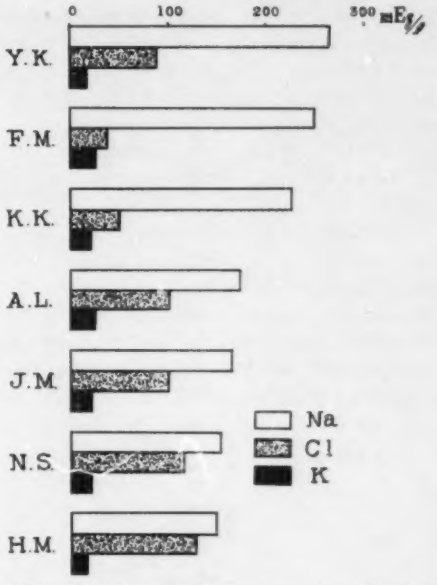

Fig. 10.—Amount of electrolytes in gallbladder bile of each case with cholesterol stone.

In cases of chronic cholecystitis without calculi, the chlorine ion showed less than 100 mEq/L.; yet it exceeded normal value, and the drying test showed no crystals or atypical ones. In cases of acute cholecystitis without calculi, the chlorine ion was more than 100 mEq/L., which was similar to that of "bilirubin stone," and the drying test showed typical crystals. This seems to indicate that crystallization by the drying test is closely related to a function of the gallbladder, in other words, the composition of bile elements rather than the presence of calculus.

Therefore, the composition of various bile elements was investigated.

Bile Acids.—The total amount of bile acids was 70 to 100 mg/cc. in the normal

state and in chronic cholecystitis, while it was less than 20 mg/cc. in cases of cholecystitis with calculi and the drying test showed typical crystals. In some cases of "cholesterol stone," however, the amount of bile acids exceeded its average value and the test showed negative. In cases of acute cholecystitis, the amount of bile acids decreased as seen in cholecystitis with calculi, and the drying test showed typical crystals as it decreased. This fact also indicates the test is related to the function of the gallbladder and its inflammatory process.

Lipoids.—Lipoids, which comprise 25% of the total solids in bile, were then investigated.

Phospholipid was extracted with alcohol-chloroform (3:1), and the extract was mineralized with perchloric acid. The amount of phosphorus was measured by the modified Allen method.[1] Assuming all phospholipid in bile to be lecithin, the amount of phosphorus ×25 was determined as that of phospholipid. As shown in Figure 11, crystallization was negative when phospholipid was more than 1,500 mg. per deciliter (1,500 mg/dl.), typical when less than 800 mg/dl., and atypical when it ranged between 1,500 and 800 mg/dl.

Cholesterol in bile was extracted with alcohol-chloroform (3:1) and the amount was measured by means of a colorimeter by the Grigault method as modified by Polonovski,[18] applying the Liebermann-Burchard reaction. As shown in Fig. 12, setting the dividing line at 200 to 100 mg/dl., crystallization was rarely observed in cases in which cholesterol was more than 200 mg/dl.; typical crystals were observed when it was less than 100 mg/dl., and crystals were negative or atypical when it varied between 200 and 100 mg/dl.

Comparing these results with the presence of calculi, the amounts of phospholipid and cholesterol were markedly decreased and typical crystals were formed in cases of cholecystitis with calculi, especially of "bilirubin stones." However, there were

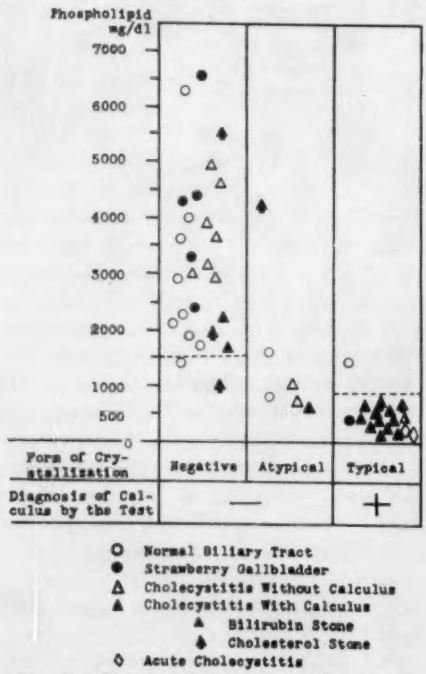

Fig. 11.—Relationship between drying test and phospholipid.

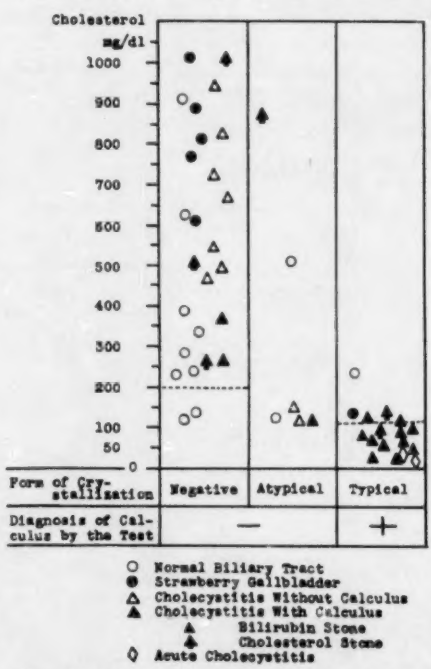

Fig. 12.—Relationship between drying test and cholesterol.

some cases of cholecystitis with calculi, both "cholesterol" and "bilirubin stones," in which typical crystals were not formed. The phospholipid and cholesterol were not decreased in these cases. On the other hand, there were some cases of acute cholecystitis in which typical crystals were formed, and the amount of lipoids was found markedly decreased in these cases. These results are similar to the relationship between the amount of total bile acids and various forms of crystallization.

Bile, which has a nature similar to that of a colloid, is capable of holding cholesterol and other insoluble substances in solution, because bile salts, lipoids, and proteins in bile act as protective colloids. These protective colloids have surface activity and they have, in their molecular structure, a hydrophilic group and a lipophilic group, and stabilize colloidal dispersion, closely binding water molecules with the strong hydrophilic group, while bile salts, making a micelle in water, play a role in holding cholesterol in solution with other protective colloids in bile.

The reason that a hydrophilic colloid is stable is because of the direct water-binding activity of its strong hydrophilic group. To study its bound water is, therefore, the most suitable index to determine the stability of the colloidal dispersion in bile.

Bound Water.—The bound water in bile was measured by the Kimoto-Ishizawa vapor pressure method[9] and Kuroda's method for micromeasurement of bound water.[10] The results were as shown in Figure 13. It appears certain that the amount of bound water is increased as the structure of crystals shifts from negative to atypical and then typical.

Total Solid.—The relationship between the drying test and the amount of total solid in bile was as shown in Figure 14. The form of crystals shifts from atypical to typical as the amount of total solid decreases, which is contrary to the relationship to the bound water.

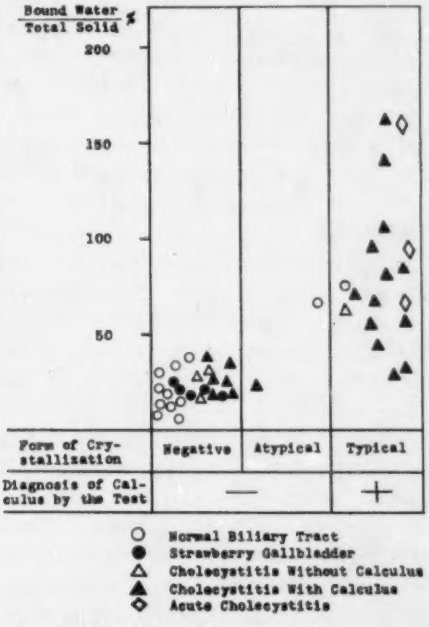

Fig. 13.—Relationship between drying test and bound water.

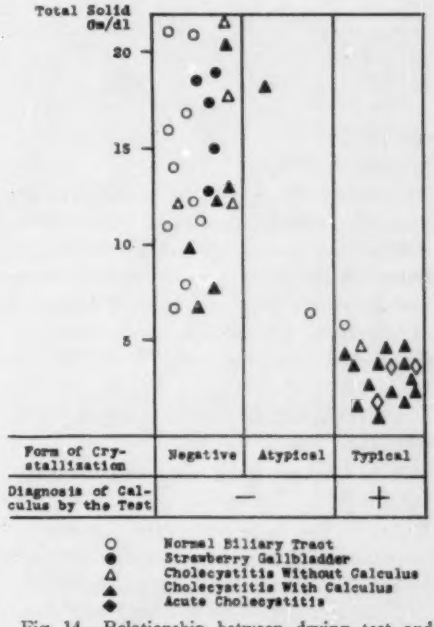

Fig. 14.—Relationship between drying test and total solid.

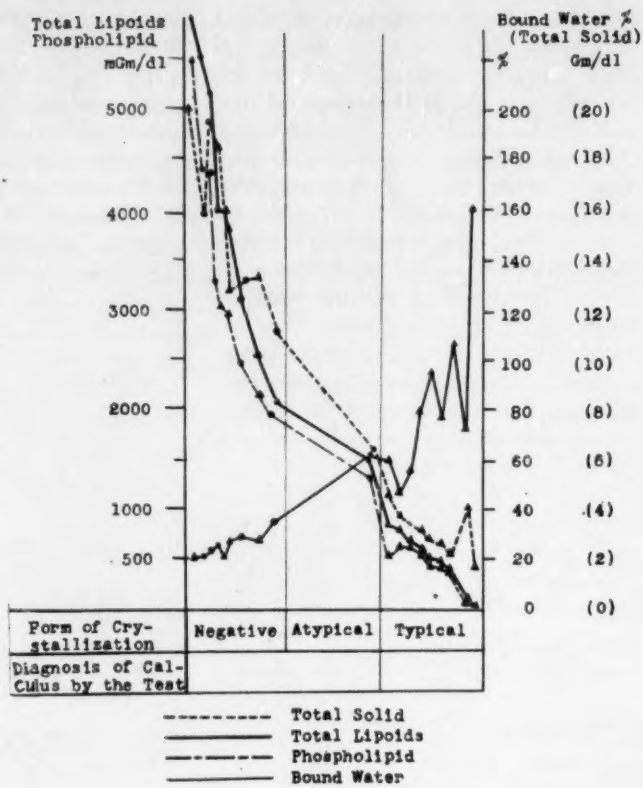

Fig. 15.—Relationship between drying test, lipoids, and bound water.

The relationships between the drying test and bile elements described above are summarized in Figure 15.

Sodium chloride, among all bile elements, holds the largest amount of bound water, and the bound water in bile in cholelithiasis is mostly due to sodium chloride. As described above, solid elements in bile, such as bile acids, phospholipid, and cholesterol, decrease in pathologic bile and Cl^- relatively increases, while Na^+ and K^+ decrease, and sodium chloride increases concomitantly with these changes in bile.

These results agree with the reports by Ravdin,[19,20] Johnston,[7,8] and Austin and Riegel,[21] that bile acids and lipoids are concentrated in normal gallbladder bile, and, when inflammation exists in the gallbladder, Cl^- ions increase while bile acid and lipoids decrease. The studies on changes of bile elements show that the drying test indicates the grade of change of bile elements, that is, the function of the gallbladder or the grade of inflammation, rather than the presence of calculus.

Since lipoid comprises 25% of solid elements in bile, and 80% of it is composed of lecithin, which has surface activity, lipoid is one of the factors to interfere with the crystallization. The presence of bile acid, however, is indispensable to bile in holding lipoid in solution. Bile acid also has strong surface activity, and forms a micelle, lecithin-bile-salt system, which was described by Isaksson.[6] It appears certain that these substances are factors interfering with the crystallization, which is dominated by a quantitative relation of bile acid and lipoid to sodium chloride in bile.

TABLE 6.—*Clinical Results*

	Total		No. of Cases	Drying Test		
				Typical	Atypical	Negative
Cholecystitis without stone	56	B bile (+)	28	16	3	15
		B bile (±)	15	8	5	2
		B bile (—)	13	9	2	2
Gallbladder stone	43	B bile (+)	15	12	0	3
		B bile (±)	8	5	1	2
		B bile (—)	20	16	2	2
Common duct stone	40	B bile (+)	8	8	0	0
		B bile (±)	16	14	0	2
		B bile (—)	16	15	0	1

An increase of bound water indicates instability of the colloidal state in bile, and the state of various bile elements is assumed to be a measure of the bound water.

Experimental Model Test

The experimental drying test was performed with various concentrations of bile acid, lecithin, and sodium chloride. A series of various volumes (0.1, 0.2, 0.3 . . . cc.) of sodium cholate solution (4 mg/0.1 cc.) was added to saline containing 100 mEq/L. of sodium chloride. A typical crystal of fern-like structure appeared upon the addition of 8 mg.; it became smaller as the amount of cholic acid increased, and disappeared when cholic acid exceeded 52 mg. When sodium deoxycholate was used, only a crystal nucleus appeared and it became smaller as the amount of sodium deoxycholate increased A part of the crystals remained in vacuoles and began to disappear where sodium deoxycholate was present, and then completely disappeared when it exceeded 64 mg.

In the case of lecithin in sodium cholate solution (1.41 mg/0.1 cc.) both a crystal nucleus and a fern-like structure appear; they become smaller as the amount of the solution increases, the fern-like structure disappears at 0.6 cc. (lecithin 8.64 mg., sodium cholate 24 mg.), and the crystal nucleus disappears at 1.0 cc.

Cholesterol in sodium cholate solution acts no differently from sodium cholate alone, and cholesterol seems to play no part interfering with the crystallization.

Clinical Evaluation of the Drying Test

The results of the test in my department with bile obtained through a duodenal tube are shown in Table 6. There were some cases of failure of diagnosis by this test in cases of cholecystitis without calculus in which "B" bile was not obtained or distinguished from other bile fractions, but good results were obtained in cases in which "B" bile was obtained.

It became clear that the crystallization indicates a state of gallbladder function, and it may show negative, even in cases of cholecystitis with calculi, if the inflammation is mild and the function of the gallbladder is well maintained. In such cases, the calculi will likely be shown by x-ray examination. As shown in Tables 7A and 7B, in 46 cases of "bilirubin stone,"

TABLE 7A.—*Cases of Bilirubin Stone in Which the Drying Test Was Typical: Forty-One Cases*

Cases	Visualization of Gallbladder by Cholangiography	Visualization of Stone by Cholangiography	Cl, mEq/L.
39 cases	—	—	Increased
H. S.	—	+	127.9
G. H.	+	—	

TABLE 7B.—*Cases of Bilirubin Stone in Which the Drying Test was Atypical: Five Cases*

Cases	Visualization of Gallbladder by Cholangiography	Visualization of Stone by Cholangiography	Cl, mEq/L.
L. S.	+	+	
R. T.	+	+	
O. N.	—	+	29.2
S. H.	+	+	15.3
T. M.	+	+	39.6

TABLE 8A.—*Cases of Cholesterol Stone in Which the Drying Test Was Typical: Ten Cases*

Cases	Visualization of Gallbladder by Cholangiography	Visualization of Stone by Cholangiography	Cl, mEq/L.
8 cases	—	—	Increased
M. L.	+	+	100.0
T. K.	—	+	145.9

41 cases showed typical crystals. In the other five cases the crystals were atypical but a roentgenogram revealed an abnormally functioning gallbladder with calculi (Table 7B).

On the other hand, in 20 cases of "cholesterol stone" (Tables 8A and 8B), the number of cases in which the drying test was positive and the number in which it was atypical or negative were the same. Most of the cases which showed atypical crystals or which were negative revealed distinct visualization of the gallbladder and calculi by x-ray examination (Table 8B).

TABLE 8B.—*Cases of Cholesterol Stone in Which the Drying Test was Atypical or Negative: Ten Cases*

Cases	Visualization of Gallbladder by Cholangiography	Visualization of Stone by Cholangiography	Cl, mEq/L.
S. F.	+	+	92.4
K. N.	+	+	58.5
T. S.	+	+	97.2
R. K.	+	+	53.0
H. W.	+	—	97.1
T. M.	+	—	21.4
S. Y.	—	+	65.3
E. K.	—	—	30.1
K. M.	—	—	98.0
A. K.	—	—	29.2

Conclusion

The special forms of crystallization by this test are not directly related to the presence of calculi, but chiefly to the function of the gallbladder or the degree of its inflammation. Since the function of the gallbladder is usually impaired in cases of cholelithiasis, especially of "bilirubin stone," which frequently shows severe inflammation, this test is far more effective as a diagnostic aid in the presence of biliary calculi than any other customary test, particularly in Japan, where "bilirubin stone" dominates (Table 9), and, if "B" bile can be obtained, this test is the best method to indicate the functional and the degree of inflammation of the gallbladder.

I wish to express my gratitude for the assistance and cooperation of Drs. Tokiro Kuwatsuka, Yoshio Nishimura, Nobuyuki Watanabe, Noriyuki Tamesue, Sunao Yoshimuta, and Yoshihiko Nakamura, who contributed to this paper.

Department of Surgery, Kyushu University.

TABLE 9.—*Frequency of Cholesterol and Bilirubin Stones Reported in Japan*

	No. of Cases	Cholesterol Stone: Pure Cholesterol Stone	Cholesterol Stone: Cholesterol Pigment Stone	Cholesterol Stone: Mixed Stone	Bilirubin Stone: Bilirubin Calcium Stone	Bilirubin Stone: Parasite Stone	Bilirubin Stone: Inorganic Stone	Proportion of Bilirubin to Cholesterol Stone
Miyake	353	4	67	7	330	45		4.8
Shiota	106	17	36	7	27	9	20	0.6
Hashimoto	160	19	44	14	45	9	29	0.7
Honda	66	1	9	4	51	1		3.7
Mutō	90	37			53			1.4
Nishimura	197	7	33	10	111	30	6	2.8
Maki	160	2	17	1	67	13		4.0
Matsukura	182	12	65	21	67	6	11	0.74
Miyake	328	6	93	6	221		2	2.19
Gallbladder stone	137	4	65	5	61		2	0.82
Common duct stone	191	2	28	1	160			5.16
Kameda	55		40		15			0.37

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Impending Cecal Perforation Secondary to a Crushing Injury of the Pelvis

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The purpose of this article is to report a case of adynamic ileus of such severity that the alarming distention of the cecum induced us to perform a cecostomy. The operative findings, substantiated by the final pathologic report, would indicate that, without surgical intervention, perforation of the cecum, with its alarming high mortality, would assuredly have occurred. Roentgenographic studies relating the greatest cecal diameter to impending cecal perforation pertain to ileus secondary to obstructing lesions of the colon distal to the cecum. In the cases reviewed by Davis and Lowman, 9 cm. is cited as the critical dimension beyond which cecal perforation was liable to occur. Eckman, Wenzke, and Abramson have recently reported a case of adynamic ileus in an elderly woman resulting in cecal perforation and her death. The greatest cecal diameter in their case was 9 cm. Their review of the recent literature indicated no other case reports of this happening in association with an adynamic ileus.

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Fig. 1.—Pelvis (AP) on admission, showing sacral and pubic rami fractures.

Report of Case

Oct. 30, 1958, a 41-year-old male truck driver was brought the the Harrisburg Hospital dispensary after being crushed between a turnpike toll booth and a house trailer. Pertinent physical findings related only to the pelvic region. Areas of ecchymosis were present on both the hips and buttocks. The abdomen was soft, and showed no distention or tenderness. X-rays revealed comminuted fractures involving both rami of the left pubis, linear fractures of both rami of the right pubis, and a fracture through the third segment of the sacrum. The hemoglobin on admission was 13.1 gm.; hematocrit 44%. The urine showed occasional RBC's.

The patient's abdomen became progressively distended. On Nov. 4 faint bowel sounds were still

Fig. 2.—Flat plate of abdomen on admission, showing no abnormalities.

audible. A flat plate of the abdomen taken Nov. 5 showed a alarming gaseous distention of the entire colon. The greatest diameter of the cecum was 14.0 cm. The air column reached to the rectum. The small bowel contained some gas. On Nov. 6 the hemoglobin was 11.2 gm.; hematocrit 36.5%; WBC 16,000, with 85% polymorphonuclears. The abdomen was tensely distended. A repeat flat plate showed the greatest cecal diameter to be 15.0 cm. The patient was toxic and dyspneic. He was taken to the operating room on Nov. 6, and under local anesthesia a cecostomy was performed by C.E.M. through a modified McBurney type incision. A 6×5 cm. area on the caput of the cecum showed hemorrhagic infarction with patchy areas of actual gangrene (histopathologic report). This gangrenous portion on the cecum was excised. The peritoneal cavity was thoroughly lavaged with dilute benzalkonium (Zephiran) solution, and a metal Babcock sump drain was placed into the pelvis through a small stab wound below the incision. The edges of the cecal defect were sutured to the skin to form a large cecostomy.

Twenty-four hours postoperatively anticoagulant therapy with warfarin was begun, as prophylaxis against venous thrombus and embolism secondary to the pelvic fractures, and bed rest. On Nov. 10, the fourth postoperative day, the patient began to pass flatus through the cecostomy, and oral fluids were allowed. On this day the hemoglobin was 10.4 gm.; hematocrit 47%; the serum bilirubin 7.0 mg. %.

On Nov. 12, the sixth postoperative day, the patient had a bowel movement from the rectum and was put on a soft diet. The Babcock drain and Foley catheter were removed. The hemoglobin was 9.9 gm.; bilirubin 5.2 mg. %. The patient was given 250 cc. of packed cells on two successive days, the hemoglobin rising to 14.0 gm. The patient was kept on chloramphenicol (Chloromycetin) until Nov. 15, two days after he had become afebrile. It was not until Nov. 19 that the abdomen became normally soft and flat. Sigmoidoscopy to 8 in. was negative. X-rays of the pelvis and a barium colon enema were performed on Dec. 1, 1958. The result of the barium enema was normal. The right pubic fractures were healed and those on the left were healing in good position. The bilirubin had returned to 0.9 mg. %. The patient was allowed out of bed in a chair.

On Dec. 8 the patient was started on oral succinylsulfathiazole (Sulfasuxidine) as part of the bowel preparation preceding planned closure of the cecostomy. Two days later he developed fever, malaise, diarrhea, hives, and auricular fibrillation. Digitilization was required. The patient then revealed a history of sensitivity to sulfonamides. On Dec. 23 the patient was returned to the operating room for closure of the cecostomy. Multiple small loculated collections of fluid were found between matted loops of small bowel in the right lower quadrant. A small wound abscess was evacuated on the sixth postoperative day. The patient was having normal bowel movements at this time. A

Fig. 3.—Flat plate done on sixth day after admission, showing marked cecal distention.

Fig. 4.—Barium colon enema done three weeks postoperatively, showing no abnormalities.

week later he was discharged as ambulatory, gaining strength daily. The wound was healed solidly.

Comment

Reflecting the extent of soft and bony tissue injury in this man was the appearance of clinical jaundice and a serum bilirubin of 7 mg. %. Necessarily the amount of hemorrhage into the soft tissue must have been considerable. The colon was severely affected, the small bowel showing a minimal distention. It is felt that the pathogenesis of this man's ileus can be more specifically pinpointed than by merely stating it followed pelvic fractures with retroperitoneal hematoma formation. It is suggested that involvement of the parasympathetic sacral outflow (S 2,3,4) by hemorrhage secondary to the sacral fracture probably produced a direct neurogenic obstruction of the left side of the colon (the vagus nerve innervates the colon as far distally as the middle of the transverse colon).

For six days following injury the patient had been becoming progressively more tensely distended, dyspneic, and toxic. The WBC rose to 16,000, with a shift to the left. Marked tenderness was present in the right lower quadrant, and generalized abdominal pain was present. The greatest cecal diameter measured 15 cm. The clinical and x-ray pictures suggested impending perforation, and the patient was, therefore, operated on. The operative findings of patchy gangrene on an area of the cecum showing a picture of hemorrhagic infarction and early peritonitis would indicate that perforation into the free peritoneal cavity was surely imminent.

This case then represents the first of its kind to be reported in the literature. The mortality and morbidity of prophylactic cecostomy compared with the high mortality of cecal perforation is minimal. It is urged, therefore, that those cases of adynamic ileus which present with progression over several days and show alarming cecal distention roentgenologically be given serious consideration for operative decompression of the vulnerable cecum. With the great increase of high speed auto travel one can expect to see cases of this nature with increasing frequency.

Summary

A case in which impending cecal perforation due to a crushing injury of the pelvis was recognized and treated by cecostomy with complete recovery of the patient is presented.

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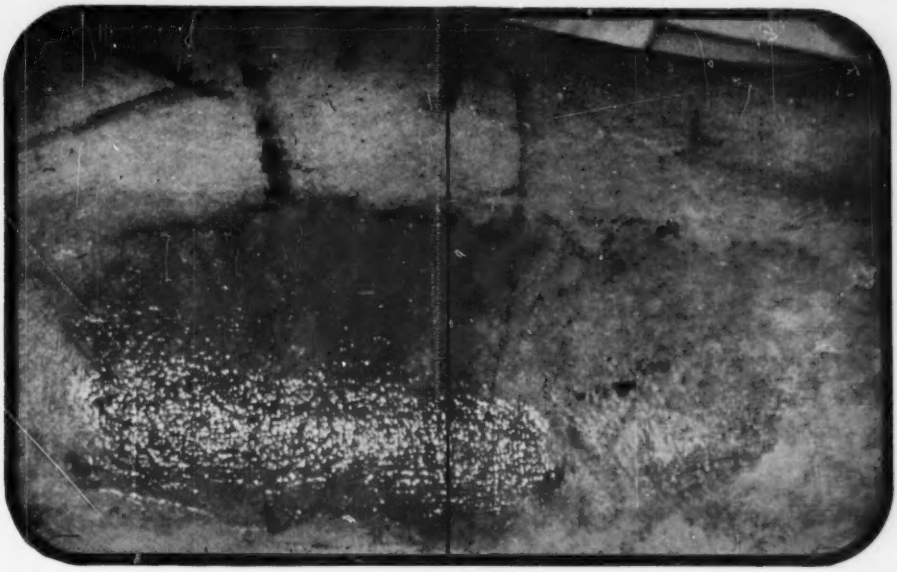

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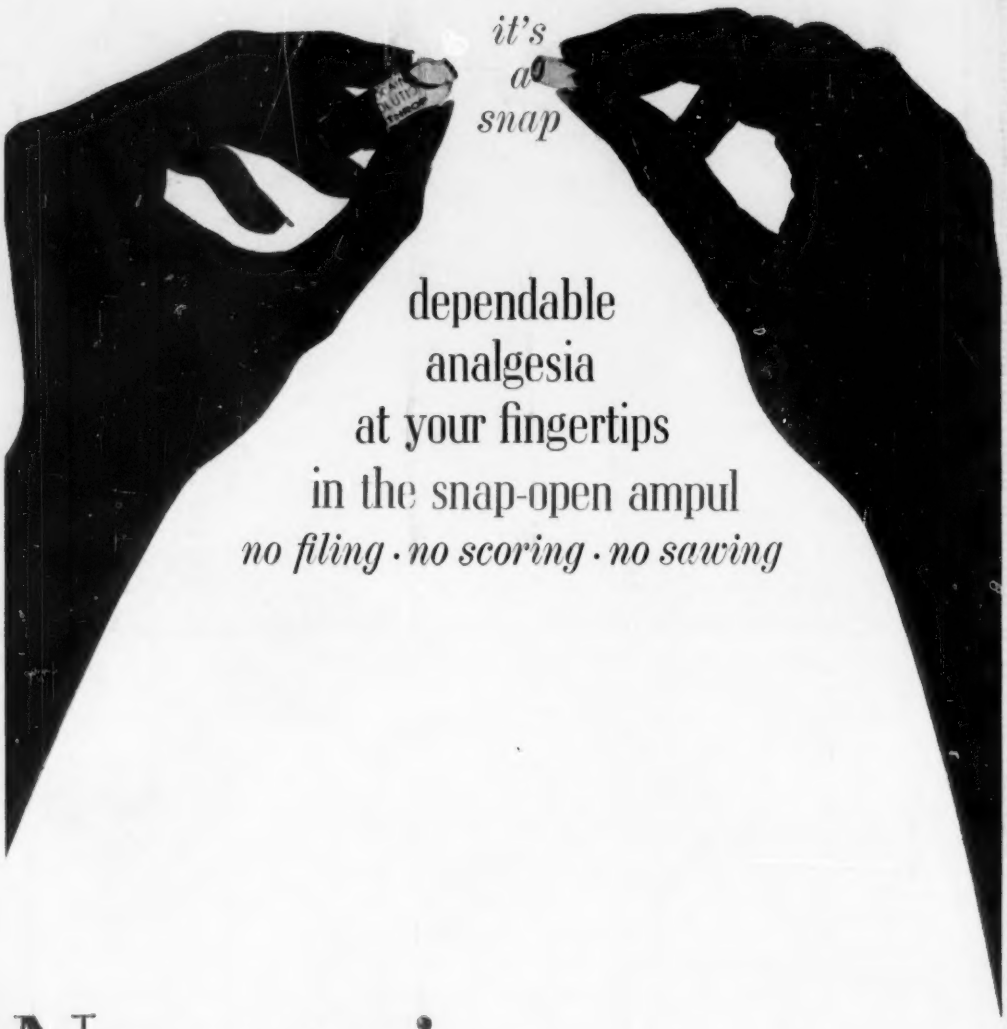

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